

363.7

5736

IDENTIFICATION OF TOXIC SUBSTANCES IN THE UPPER ILLINOIS RIVER



ENR
*Illinois Department of
Energy and Natural Resources*

Jim Edgar, Governor
John S. Moore, Director



John S. Moore, Director

ERRATA

Identification of Toxic Substances in the Upper Illinois River

ILENR/RE-WR-92/07

Dr. Frank S. Dillon was the Project Manager for this research endeavor, and made a substantial creative contribution to the drafting of the final report. His efforts were left unacknowledged inadvertently.

A corrected title page and NTIS form are attached. ENR regrets the error.

ILENR/RE-WR-92/07
Printed: October 1992
Reprinted: November 1992
Contract: WR 3691
Project: 89/215

Identification of Toxic Substances in the Upper Illinois River

Final Report

Prepared by:

River Research Laboratory
Forbes Biological Station
Illinois Natural History Survey
P.O. Box 599
Havana, IL 62644

Principal Investigators:

Richard E. Sparks
Philippe E. Ross

Project Manager:

Frank S. Dillon

Prepared for:

Illinois Department of
Energy and Natural Resources
Office of Research and Planning
325 W. Adams, Room 300
Springfield, IL 62704-1892

Jim Edgar, Governor
State of Illinois

John S. Moore, Director
Illinois Department of
Energy and Natural Resources

REPORT DOCUMENTATION PAGE		1. REPORT NO. ILENR/RE-WR-92/07	2.	3. Recipient's Accession No.
4. Title and Subtitle Identification of Toxic Substances in the Upper Illinois River		5. Report Date October 1992		
7. Author(s) Richard E. Sparks , Philippe E. Ross and Frank S. Dillon		6. Performing Organization Rept. No.		
9. Performing Organization Name and Address River Research Laboratory Forbes Biological Station Illinois Natural History Survey P.O. Box 599 Havana, IL 62644		10. Project/Task/Work Unit No. 89/215		
12. Sponsoring Organization Name and Address Illinois Department of Energy and Natural Resources Office of Research and Planning 325 West Adams Street Springfield, IL 62704-1892		11. Contract(C) or Grant(G) No. (C) (G) WR3691		
15. Supplementary Notes		13. Type of Report & Period Covered		
		14.		

16. Abstract (Limit: 200 words)

Between 1955 and 1958, several abundant species of aquatic insects, snails and finger-nail clams practically disappeared from the upper Illinois River. These declines had equally drastic repercussions on the populations of ducks and fish that fed upon these invertebrates. The situation changed very little into the 1980s, despite improvements in water quality. This research found that porewater from relevant river sediments contains a toxic factor that inhibits the filtering ability of fingernail clams and also negatively effects the water flea, while stimulating alga and bacteria growth. All evidence points to ammonia as the toxic agent. Also, at two sites, the porewater contained toxic oil products, including polycyclic aromatic hydrocarbons, such as naphthalene. A three phase Toxicity Identification and Evaluation methodology was utilized in reaching these conclusions.

17. Document Analysis a. Descriptors

Water Pollution, Toxicity, Water Pollution Effects (Animals),
Sediments, Sedimentation, Suspended Sediments

b. Identifiers/Open-Ended Terms

Sedimentation - Illinois River
Toxicity - Illinois River
Water Pollution - Illinois River

c. COSATI Field/Group Biological and Medical Sciences; Environmental Biology

18. Availability Statement No restriction on distribution. Available at IL Depository Libraries or from National Technical Information Service, Springfield, VA	19. Security Class (This Report) Unclassified	21. No. of Pages 72
	20. Security Class (This Page) Unclassified	22. Price

ILENR/RE-WR-92/07
Printed: October 1992
Reprinted: November 1992
Contract: WR 3691
Project: 89/215

Identification of Toxic Substances in the Upper Illinois River

Final Report

Prepared by:

River Research Laboratory
Forbes Biological Station
Illinois Natural History Survey
P.O. Box 599
Havana, IL. 62644

Principal Investigators:

Richard E. Sparks
Philippe E. Ross

Prepared for:

Illinois Department of
Energy and Natural Resources
Office of Research and Planning
325 W. Adams, Room 300
Springfield, IL 62704-1892

Jim Edgar, Governor
State of Illinois

John S. Moore, Director
Illinois Department of
Energy and Natural Resources

NOTE

This report has been reviewed by the Illinois Department of Energy and Natural Resources (ENR) and approved for publication. Statements made by the author may or may not represent the views of the Department. Additional copies of this report are available through the ENR Clearinghouse at 800/252-8955 (within Illinois) or 217/785-2800 (outside Illinois).

Printed by the Authority of the State of Illinois.

Date Printed:	October 1992 Reprinted: November 1992
Quantity Printed:	250 200 reprints
Referenced Printing Order:	

One of a series of research publications published since 1975. This series includes the following categories and are color coded as follows:

Energy Resources	-RE-ER	-Red
Water Resources	-RE-WR	-Blue
Air Quality	-RE-AQ	-Green
Environmental Health	-RE-EH	-Grey
Insect Pests	-RE-IP	-Purple
Information Services	-RE-IS	-Yellow
Economic Analysis	-RE-EA	-Brown

Illinois Department of Energy and Natural Resources
Office of Research and Planning
325 West Adams, Room 300
Springfield, Illinois 62704-1892
217/785-2800

ACKNOWLEDGEMENTS

This research was administered and partially funded by the Illinois Department of Energy and Natural Resources (IDENR), with the balance of funding from the Illinois Environmental Protection Trust Fund. The development of the fingernail clam bioassay, one of five bioassays used in this project, was funded separately by grant F-94-R from the Federal Aid in Fish Restoration Program (Wallop-Breaux Act), administered by the Illinois Department of Conservation.

Many people contributed to this project. Dr. Anthony A. Paparo, Department of Zoology and School of Medicine, Southern Illinois University at Carbondale, did much of the early development of a variety of methods for assessing responses of clams and mussels to contaminants. Ms. Diane Dillon and Mr. Jeffrey Arnold provided valuable technical assistance at Western Illinois University, as did Ms. Louann Burnett at the Natural History Survey in Champaign, and Mr. K. Douglas Blodgett at the Natural History Survey's Long Term Resource Monitoring Station in Havana. Ms. Camilla Smith provided secretarial assistance at the River Research Laboratory of the Stephen A. Forbes Biological Station in Havana. The research could not have been done without the laboratory facilities, office space, and equipment provided by Dr. Richard V. Anderson and the Department of Biological Sciences at Western Illinois University--to both we express our great appreciation. This project would not have come into being without the sustained interest of Dr. John Marlin, Director of the Illinois Pollution Control Board, in the mysterious die-off of fingernail clams in the Illinois River and the widespread ecological repercussions of their failure to recolonize. Finally, we thank our project officers at DENR, Ms. Linda Vogt and Mr. William Denham.

TABLE OF CONTENTS

Executive Summary.....	xi
1.0. Introduction.....	1
2.0. Project Goals and General Approach.....	9
3.0. Methods.....	11
3.1. Site Description.....	11
3.2. Sampling Design.....	12
3.2. Sample Collection Procedures.....	12
3.3. Chemical analysis.....	15
3.4. Bioassays.....	16
3.5. Toxicity identification and evaluation procedures.....	20
3.5.1. Phase I.....	20
3.5.2. Phase II.....	21
3.5.3. Phase III.....	24
4.0. Results.....	25
4.1. Relative toxicity.....	25
4.2. Toxicity Identification Evaluation - Phase I.....	29
4.2.1. 1990.....	29
4.2.2. 1991.....	32
4.3. Toxicity Identification Evaluation - Phase II.....	32
4.4. Toxicity Identification Evaluation - Phase III.....	40
4.5. Sensitivity of Recolonizing Clams.....	42
5.0. Discussion.....	45
6.0. Literature Cited.....	53

LIST OF FIGURES

Figure 1.1. Sampling stations on the Illinois Waterway.....	2
Figure 1.2. Sampling stations in the Chicago area.....	3
Figure 1.3. TIE procedures.....	7
Figure 1.4. Phase I procedures.....	8
Figure 4.1. Results of toxicity tests with five test species.....	26
Figure 4.2. Fingernail clam response to porewaters.....	28
Figure 4.3. Correlation of toxicity with ammonia.....	41

LIST OF TABLES

Table 3.1. Location of sampling stations.....	13
Table 4.1. Toxicity of porewater to <i>Ceriodaphnia dubia</i>	30
Table 4.2. Results of treating sediment porewater 1990.....	31
Table 4.3. Toxicity and ammonia concentrations.....	33
Table 4.4. Results of treating sediment porewater 1991.....	34
Table 4.5. Results of treating sediment porewater with zeolite...	35
Table 4.6. Toxicity of sediment porewater.....	37
Table 4.7. Toxicity of extracts from sediment porewater.....	38
Table 4.8. Constituents of elutriates.....	39

In a brief span of 3 years, 1955-1958, several abundant species of aquatic insects, snails, and fingernail clams practically disappeared from a 170-km reach of the Illinois River, from Hennepin on the north to the mouth of the Sangamon River on the south. The declines of the fingernail clam, *Musculium transversum*, were particularly spectacular: from average densities of 21,000 animals per square meter to zero in Peoria Lake and backwater lakes near Havana. The declines had drastic repercussions on the ducks and fish that fed upon the invertebrates. The lesser scaup duck, or bluebill, virtually stopped using the Illinois River as a major migration route, and there was a decline in the condition and growth of bottom-feeding fish, including sport fish, such as channel catfish, and commercially important species, such as common carp.

The situation changed very little into the 1980s, despite improvements in water quality (e.g., higher dissolved oxygen levels attributable to improved waste treatment in the Chicago-Joliet area and Peoria). This lack of recovery was especially puzzling because the invertebrates are capable of rapidly recolonizing barren areas; seed populations are available in spring-fed areas of Peoria Lake and in tributaries and these organisms have short, rapid life cycles.

We found that porewater from Illinois River sediments contains a toxic factor that inhibits the filtering ability of the clam, and the toxicity increases upstream, peaking near Lockport. We observed the same pattern of sediment toxicity with a different test organism, also representing a class of important food organisms for fish and waterfowl: the water flea, *Ceriodaphnia dubia*. In contrast, the porewater actually stimulates an alga and bacteria, but this is not surprising because of the great physiological differences among plants, bacteria and animals.

Toxicity greatly decreased when the porewater was made slightly more acid and porewater became nontoxic when filtered through a resin that removed ammonia. Removal of heavy metals with a chelating agent had no effect on toxicity. All the evidence points to ammonia as the culprit, especially since toxicity in all tests correlated highly with the concentration of ammonia, which is known to be toxic to aquatic animals. Since ammonia is a nutrient for plants and certain types of bacteria, the presence of ammonia likewise could explain the stimulation of these organisms.

Although ammonia appears responsible for the major upstream-downstream pattern in toxicity, there were two sites where the porewater contained visible signs of oil and the toxicity was associated with petroleum hydrocarbons, including PAHs (polycyclic aromatic hydrocarbons) such as naphthalene.

During the course of this study, several species of fingernail clams, including *M. transversum*, reappeared in the Chicago area waterways and in the Illinois River at Peoria and Havana. There are at least four possible explanations for this surprising reappearance of clams in the same general areas where the porewaters tested toxic. First, we found that clams recolonizing the upper Illinois are more resistant to ammonia than the clams from the lower Illinois, where the organisms were obtained for all of the early bioassays. Second, our previous research demonstrated that the surface layers of sediment in some areas are less toxic than layers a few centimeters deeper. Toxicity may have been overestimated in tests where surface and deep layers of sediment were mixed prior to testing. Third, toxic episodes may be brief and infrequent, allowing organisms to colonize in between episodes. Fourth, the distribution of toxicity in sediments may be extremely patchy, so that healthy organisms are found adjacent to barren areas. If the latter two hypotheses prove to be true, acute toxicity in the Illinois River has changed recently from a widespread problem to a more localized or episodic problem. Reduction of toxicity in surface sediments may reflect recent reductions in ammonia loading from sewage treatment plants in the Chicago area, although it is not clear whether the sources of ammonia in the porewaters are effluents, the deeper layers of sediments, or both.

1.0 INTRODUCTION

The quality of sediments is critical to the ecological health of aquatic ecosystems. Benthic organisms that live in sediments are key links in food chains that lead from nutrients in water and sediment to higher level consumers, such as fish and ducks. Sediments in aquatic systems can be both sinks and sources for inorganic and organic contaminants. At present, the extent of the sediment contamination problem is largely unknown. Comprehensive assessments of the accumulation of contaminants from agricultural, municipal, and industrial sources in sediments of our rivers, lakes and estuaries have not been completed. Currently, the U.S. Environmental Protection Agency has identified 134 sites with serious sediment contamination problems (USEPA 1988). In addition, 41 areas in the Great Lakes (IJC 1988), 50 coastal sites, and 85 wildlife refuges have been identified where contaminated sediments pose a problem (USEPA 1988).

In Illinois, contaminants have been identified in sediments throughout much of the Illinois River and its associated tributaries and waterways (Figures 1.1 and 1.2; Cahill and Steele 1986; Cahill and Autrey 1987; Blodgett et al. 1984; Mathis et al. 1973; Polls et al. 1985; Harrison et al. 1981; Coleman and Sanzalone 1991; Bhowmik and Demissie 1989; Sparks and Blodgett 1984; and Fitzpatrick and Bhowmik 1990). Two-thirds of the population of the state lives in the Illinois River basin which drains approximately half the state (Talkington 1991). The river historically has been one of the most productive rivers in North America in terms of fish and wildlife populations. In 1908,

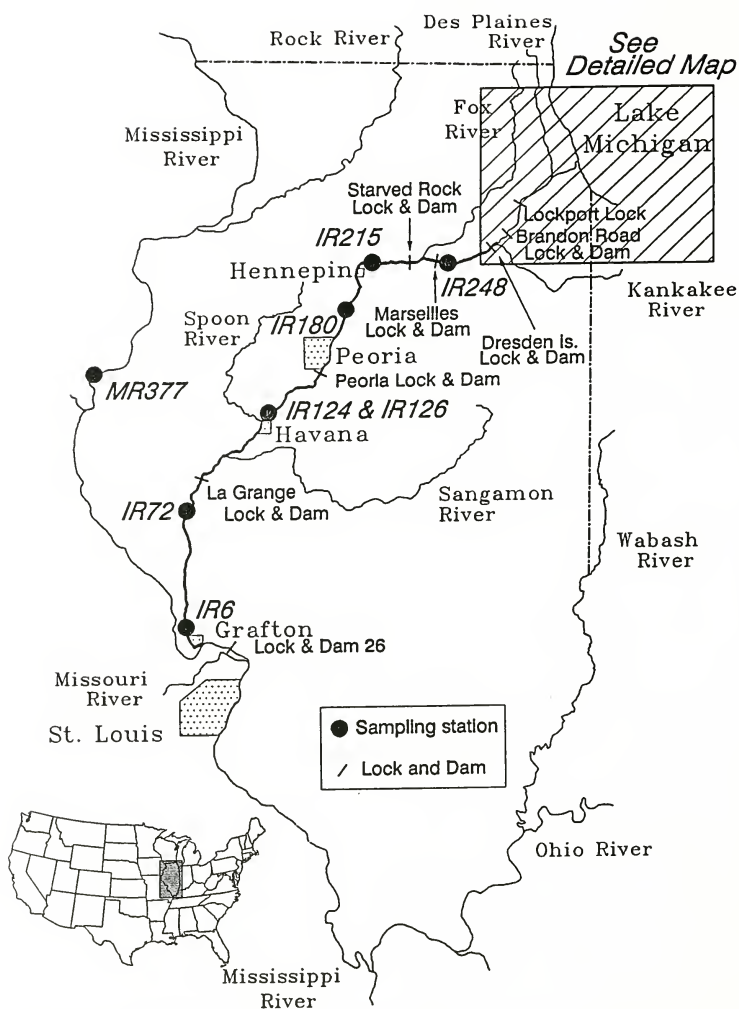


Figure 1.1. Location of sediment sampling stations on the Illinois Waterway. Stations are identified according to river miles: Illinois River miles (IR) start at Grafton at mile 0.0 and proceed upstream to Chicago. A reference station was established on the Mississippi River (MR), 377 miles above the confluence with the Ohio River.

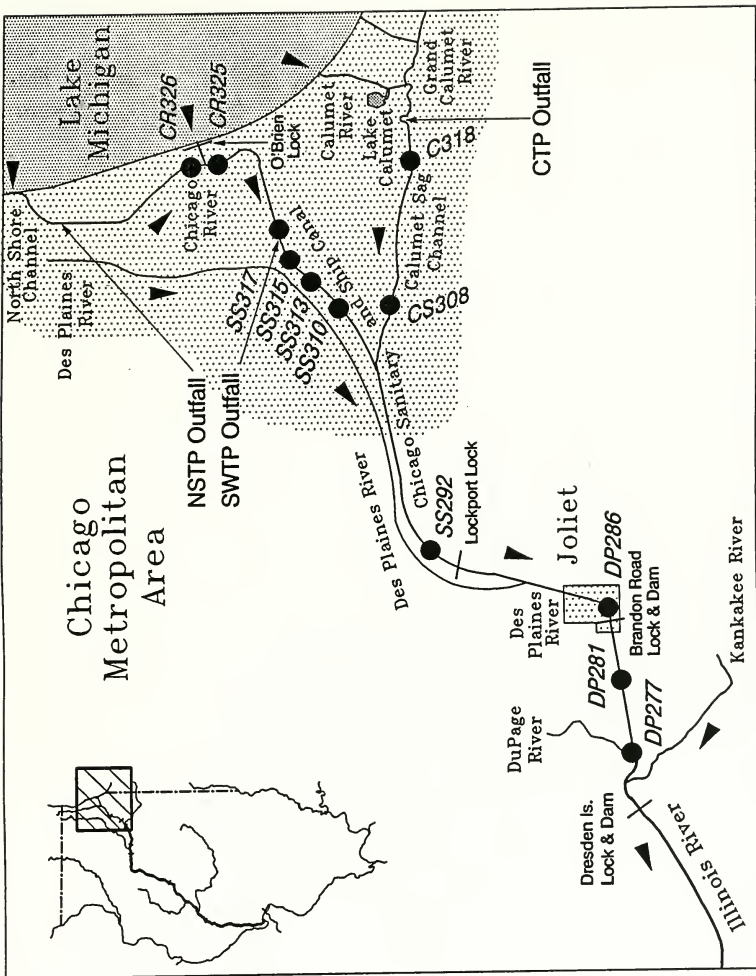


Figure 1.2. Location of sediment sampling stations in the Chicago-Joliet area. Stations (black circles) are identified according to distance (in miles) upstream from the confluence with the Mississippi River at Grafton, and according to the name of the reach. DP = Des Plaines River. SS = Chicago Sanitary and Ship Canal. CS = Calumet Sag Channel. CR = Chicago River. The location of the major sewage treatment plant outfalls in the Chicago area are noted: NSTP = northern sewage treatment plant, SWTP = southern sewage treatment plant, and CTP = Calumet treatment plant. Arrowheads indicate usual direction of flow in the waterways.

a 320-km (200-mile) reach from the great bend at Hennepin to the confluence with the Mississippi River at Grafton (Figure 1.1) produced 10% of the total U.S. harvest of freshwater fish and two thousand commercial fisherman made a living from the river (U.S. Department of Commerce and Labor 1911). The commercial yield was 24 million pounds annually, or about 178 pounds per acre of permanent water (Lubinski et al. 1981). By the 1950s the yield had dropped to 38 pounds per acre; since the 1970s the yield has been a low 4 pounds per acre, totaling only 0.32% of the total U.S. catch (Sparks 1984). Similar downward trends were recorded over the same period for two other indicators of biological productivity: waterfowl and sport fish populations (Bellrose et al. 1979; Sparks 1977; Sparks 1992). Major commercial fish species and the diving ducks feed on bottom-dwelling invertebrates such as clams, snails, aquatic worms, and aquatic insects. In the early 1900's a healthy benthic community contributed to the tremendous production of fish and waterfowl. A major component of that benthic community was a small clam, *Musculium transversum* (Family Sphaeriidae). Now, this clam as well as other small mollusks, mayflies, midges, and other burrowing aquatic insects has been virtually eliminated from certain reaches of the Illinois River (Starrett 1972, Anderson 1977, Sparks et al. 1986).

Declines in the benthic invertebrates of the Illinois River system have been linked to sediment-associated toxicity (Sparks et al. 1981; Blodgett et al. 1983; Sparks 1984). Aquatic sediments can accumulate both inorganic and organic chemicals that are absorbed to particulate matter or are in solution in sediment porewater (Salomons et al. 1987, Tessier and Campbell 1987). Porewater (also called interstitial water) is the water occupying the spaces between the sediment particles. These

contaminants can have acute toxic effects on benthic organisms, or accumulate slowly in the organisms until some toxic threshold is reached.

The toxicity to aquatic organisms is known for only a fraction (<1%) of the approximately 50,000 compounds manufactured in the U.S. (Martell et al. 1990). This situation is further complicated by the fact that organisms usually are simultaneously exposed to a number of chemicals (Giesy et al. 1990). The toxic responses associated with these mixtures of compounds depends on their bioavailability--some contaminants are bound to sediment particles or otherwise unavailable to organisms. For instance, the bioavailability of non-ionic organic compounds depends on the total organic carbon content (TOC) of the sediment (Nebeker et al. 1989, Swartz et al. 1990, Di Toro et al. 1991) and the bioavailability of certain cationic metals depends on the acid-volatile sulfide (AVS) content of the sediment (Di Toro et al. 1990, Ankley et al. 1991, Carlson et al. 1991). Due to the complex mixtures of contaminants present in most toxic sediments, as well as the effects that sediment matrices may have on the bioavailability of compounds, it has been difficult to link specific compounds with toxicity. The traditional approach to identifying toxic agents has been to correlate toxicity with the concentrations of chemicals in the bulk sediment sample (Carr et al. 1989). This approach does not work well with complex mixtures and does not address the question of bioavailability. The dose response curve for biological effects from certain chemicals is not correlated to the bulk sediment concentration but rather to the porewater concentration (Di Toro et al. 1991). The recent development of Toxicity Identification and Evaluation (TIE) methodology has made it possible to iden-

tify specific toxic compounds in complex mixtures (Figure 1.3; Mount and Anderson-Carnahan 1988; Mount and Anderson-Carnahan 1989; Mount 1988).

TIE procedures use toxicity-based fractionation schemes to characterize and identify compounds in aqueous samples that exhibit toxicity to aquatic organisms. Although TIE cannot be used on bulk sediments, it can be applied to the aqueous fraction (porewater). Previous studies (Adams et al. 1985; Swartz et al. 1985; Knezovich and Harrison 1988; Connell et al. 1988; Swartz et al. 1988, Di Toro et al. 1992) have shown a correlation between toxicity or bioaccumulation of a number of contaminants by benthic macroinvertebrates, on the one hand, and porewater concentrations on the other. The TIE procedures are designed to address multiple toxicant interactions as well as matrix effects on bioavailability. The major strength of TIE is that it allows direct relationships to be established between toxicity and chemical analyses. TIE is a phased approach that is designed to isolate, identify and confirm the presence of acutely toxic compounds. TIE methodology for identification of chronically toxic compounds is currently under development (USEPA 1992). Phase I of TIE consists of a series of chemical and physical manipulations designed to remove or render biologically unavailable generic classes of compounds (Figure 1.4). Phase II uses information from Phase I to focus appropriate analytical methods on toxic fractions. Phase III consists of methods designed to verify that the suspected toxicant is the actual toxicant. TIE methodology has been applied to sediments from the Great Lakes (Ankley et al. 1990) and the Calumet Sag Channel of the Illinois River system (Schubauer-Berigan and Ankley 1991). We applied these techniques to sediments from the Illinois River System in an effort to identify the substance or substances responsible for the declines of the benthic invertebrates.

TOXICITY-BASED TOXICITY IDENTIFICATION EVALUATION

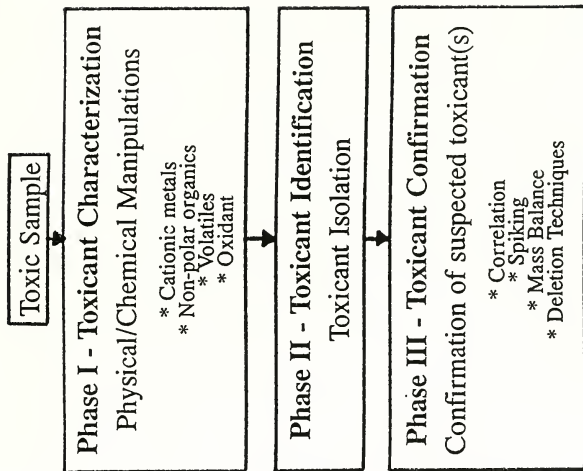


Figure 1.3. The three phases of Toxicity Identification and Evaluation (TIE) procedures.

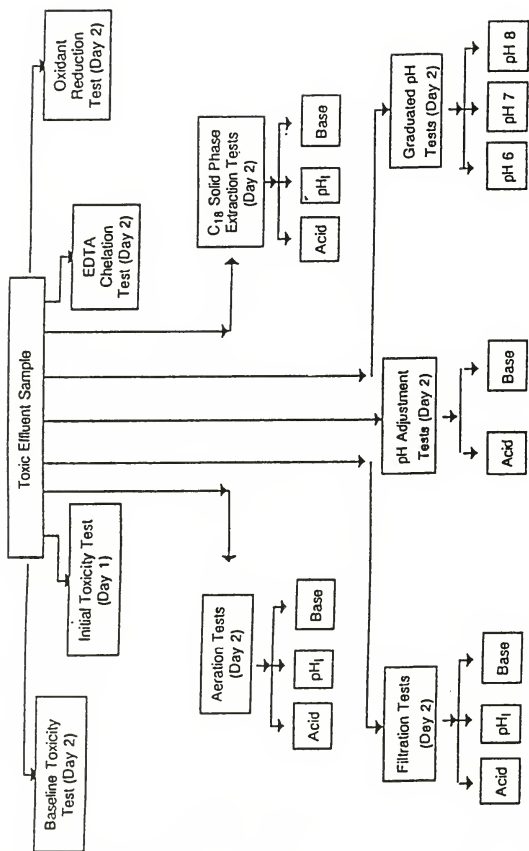


Figure 1.4. Steps involved in Phase I Toxicity Identification and Evaluation (TIE). Source: Mount and Anderson-Carnahan 1988.

2.0 PROJECT GOALS AND GENERAL APPROACH

The primary goal was to identify the toxicants in the sediments of the Illinois Waterway. In addition, we hoped to identify upstream-downstream patterns in toxicity and follow toxicity gradients upstream to sources.

We tested the toxicity of sediments taken widely along the entire length of the Illinois Waterway, and from one reference site on the Upper Mississippi River (Figures 1.1 and 1.2). Next, sediments that exhibited toxicity were subjected to Phase I Toxicity Identification and Evaluation (TIE) procedures (pH adjustment, addition of a chelating agent, etc.), to characterize the toxicants (see Figures 1.3 and 1.4). If the toxicity at all sites varied the same way in response to the Phase I treatments, we would know we were dealing with one class of toxicants, or perhaps even one major toxicant, and we could focus additional sampling on the most toxic reach in the hope of identifying the major source. At the same time, Phase II and Phase III TIE procedures would confirm the identity of the toxicant or at least narrow the range of suspect chemicals. However, if Phase I testing indicated that different classes of toxicants occurred in different reaches of the waterway, then we would have a much more complex task of identifying multiple toxicants and multiple sources--a task that might extend well beyond the budget and time limitations of this research.

3.0 METHODS

3.1 Site Description

Today's Illinois Waterway is approximately 327 miles (526 km) long connecting Lake Michigan and the Chicago-Joliet metropolitan area with the Mississippi River and the agricultural heartland, near Grafton, Illinois (Figure 1.1). The headwaters are in the highly industrialized Chicago area where the flow of the Chicago River was reversed to carry wastes away from Lake Michigan into the Illinois River via the Chicago Sanitary and Ship Canal and the downstream portion of the Des Plaines River (Figure 1.2). The Calumet Sag Channel enters the Sanitary and Ship Canal near Lemont. The Illinois River proper begins with the confluence of the Des Plaines and Kankakee rivers, and flows through a predominantly agricultural drainage, although the industrial city of Peoria is situated approximately mid-way along the waterway.

Locations on the waterways are designated by river mile as recorded in river charts prepared by the U.S. Army Corps of Engineers (1987) and by markers along the waterways, starting with mile 0.0 at the confluence with the Mississippi and proceeding upstream to Chicago. The following abbreviations are used in the text, figures, and tables to identify reaches of the waterway, and stations are identified by reach abbreviation and river mile:

IR	Illinois River proper
DP	Des Plaines River
CS	Calumet Sag Channel
SS	Chicago Sanitary and Ship Canal
CR	Chicago River

The one reference station on the Upper Mississippi River is located 377.0 miles above the confluence with the Ohio River and is designated MR 377.0. The locations of the sample stations are given in Table 3.1 and Figures 1.1 and 1.2. In accordance with Corps of Engineers terminology, the designation "left bank" or "right bank" assumes the observer is facing downstream.

3.2 Sampling Design

Nineteen sampling stations were established throughout the Illinois Waterway (Figures 1.1 and 1.2). Samples were collected from 15 stations from November 1989 to June 1990, and from all 19 stations from November 1990 to June 1991 (Table 3.1).

3.2 Sample Collection Procedures

It is important to limit the disruption of the sediment so that toxicity evaluations are conducted under conditions that closely match the *in situ* conditions (ASTM 1991). The most appropriate sediment sampling device is study specific. Sediment corers generally disrupt the sediment little but collect a limited sample volume (ASTM 1991). This study employed a battery of bioassays as well as the TIE procedures, all of which used sediment porewater. The volume of porewater needed for this work made the use of sediment corers impractical. We used a 25.4 cm (10-inch) Ekman dredge that works well in the soft to semi-soft sediments that characterize the Illinois Waterway and collects a relatively large sample volume (ASTM 1991).

Table 3.1. Location of sampling stations.

River Mile	Description
CR 326.4	North Branch of Chicago River at Michigan Avenue Bridge
CR 324.8	South Branch of the Chicago River at Harrison Street Bridge
CS 318.5	Upstream of Division Street Bridge on Calumet Sag Channel
CS 307.4	Upstream of 104th Street Bridge on Calumet Sag Channel
SS 317.0	-5 m from left bank
SS 315.3	-25 m from left bank
SS 313.0	-2 m from right bank downstream of Route 171 Bridge
SS 310.0	-10 m from left bank upstream from Justice Navigation Light
SS 292.2	10 m upstream of sunken barge and 30 m from right bank
DP 286.3	Left bank -300 m upstream of Brandon Road Lock and Dam
DP 281.1	-30 m from left bank across from Olin Chemical
DP 277.0	Upstream of Du Page River Daymark -500 m from right bank
IR 248.2	-100 m upstream of Ballards Island
IR 215.0	Center of Turner Lake
IR 180.0	Upper Peoria Lake, south of Chillicothe
IR 125.5	SE Corner of Lake Chautauqua
IR 72.0	Center of Meredosia Lake
IR 6.0	Entrance to Swan Lake
MR 377.0	Montrose Flats, Pool 19, Mississippi River

Note: The Illinois Waterway includes the Illinois River (IR), Des Plaines River (DP), Chicago Sanitary and Ship Canal (SS), Chicago river (CR), and Calumet Sag Channel (CS). The mileages start at IR 0.0 at the confluence with the Mississippi and proceed upstream to Chicago. Mileages on the Upper Mississippi River (MR) start at the confluence with the Ohio. "Right" and "left" assume the observer is facing downstream.
m = meters.

The sampler was rinsed with river water at the site prior to sediment collection. The sample was placed in prewashed (Biossoap wash, ultrapure water rinse) high density polyethylene containers. High density polyethylene containers are relatively inert and are optimal for samples contaminated with a variety of chemicals (ASTM 1991). The containers were filled completely to achieve zero sample head space. Sample containers were placed on ice as soon as possible following collection (never exceeding 2 hours). Samples were transported to the laboratory and stored at 4°C for no more than two weeks as recommended by Anderson et al. (1984).

We used sediment porewater in our toxicity tests. Numerous studies (Adams et al. 1985; Swartz et al. 1985; Knezovich and Harrison 1988; Connell et al. 1988; Swartz et al. 1988, Di Toro et al 1992) have shown that porewater is an appropriate surrogate for bulk sediment. Porewater can be collected from sediment samples by several methods: centrifugation, squeezing, suction, and equilibrium dialysis (ASTM 1991). Centrifugation is generally used if large volumes of porewater are required (Edmunds and Bath 1976). Constituents such as salinity, dissolved inorganic carbon, ammonia, sulfide, and sulfate are generally not affected as long as oxidation is prevented; however, dissolved organic carbon (DOC) and dimethylsulfide may be significantly reduced using this method (Howes et al. 1985). Sediment porewater was extracted by centrifugation at 4000 g (g = the acceleration due to gravity) at 4°C for 45 minutes. Sample porewater was stored with zero head space at 4°C in a decontaminated cubitainer for a maximum of 1 week. The time from collection to testing ranged from 1 to 6 days, and averaged 2.6 days for

all sediments.

Surface water samples were collected just prior to collection of sediment. Surface water was collected from approximately mid-depth in the water column using a Van Dorn sampler. Samples were placed in pre-cleaned cubitainers and immediately placed on ice. Surface water samples were stored at 4°C for a maximum of one week.

3.3 Chemical Analysis

Routine chemical measurements were taken on both surface water and porewater samples. Samples were brought to ambient temperature (20-24°C) prior to making the following measurements:

SURFACE WATER

Dissolved Oxygen
pH (negative log of the hydronium
ion concentration (minus $\{H^+\}$))
Conductivity
Alkalinity
Hardness
Total Ammonia-N (ammonia measured
as nitrogen, N)

PORE WATER

Dissolved Oxygen
pH
Conductivity
Alkalinity
Hardness
Total Ammonia-N
Total Cl (chlorine)
H₂S (hydrogen sulfide)
Sulfide

Dissolved oxygen was measured using a standard Y.S.I. Model 57 oxygen meter with a Y.S.I. Model 5739 probe. Temperature and pH were measured using a Jenco Microcomputer pH-Vision 6071 pH meter with a temperature-compensating Ross electrode. Specific conductance was measured using a Y.S.I. Model 35 Conductance Meter with a Y.S.I. Model 3401 probe. Total alkalinity was measured using the ASTM (1982) standard titration method. Total hardness was measured using the EDTA titrimetric method (APHA 1989). Total ammonia nitrogen was determined using the Nesslerization method (APHA 1989), total residual chlorine by the DPD colorimetric method (APHA 1989), sulfide by the methylene blue

method (APHA 1989) and hydrogen sulfide by the lead sulfide method. All instrumentation was calibrated prior to testing.

We intended to calculate the fraction of the total ammonia that existed in the un-ionized state during the toxicity tests (see below). In aqueous ammonia solutions an equilibrium exists between ammonia in the highly toxic un-ionized form (NH_3) and ammonia in the relatively nontoxic ionized form (NH_4^+). The dominant factor regulating the equilibrium between the two forms is pH, with the temperature having a lesser effect. We were not able to calculate un-ionized ammonia concentrations in the toxicity tests because the pH of the porewater drifted slightly during the tests. Temperature was held constant. However, our subsequent analysis of the correlation between toxicity and total ammonia is justified because the initial pHs of the samples were similar (6.5-7.25) and all drifted in a similar manner, so the un-ionized ammonia concentrations were some consistent fraction of the total ammonia concentrations in all the test chambers.

Measurements of total organic carbon (TOC) were performed on bulk sediment samples. The results are expressed in percent organic carbon.

3.4 Bioassays

Burton (1991) described several components that should be considered in selecting a bioassay for toxicity assessment:

Components of an Optimal Toxicity Assay

1. Verification components
 - Ecosystem relevance
 - Species sensitivity patterns
 - Appropriate test phase
 - Short or long exposure period
 - Definitive response dynamics

2. Resource components
 - Organism availability
 - Laboratory availability
 - Expertise required
 - Expense and time required
3. Standardization components
 - Approved standard methods
 - Reference data base
 - Interlaboratory validation
 - Quality assurance and control criteria

Verification components such as ecosystem relevance, sensitivity, and discriminatory ability are so critical that multiple species and endpoints should be incorporated in testing programs for sediment toxicity assessments, according to Burton (1991). Therefore we measured the relative toxicity of the sediment porewater with a battery of bioassays that included the following test organisms: the marine bacterium, *Photobacterium phosphoreum* (MicrotoxTM), the freshwater alga, *Selenastrum capricornutum*, the rotifer, *Branchionus calyciflorus*, the daphnid, *Ceriodaphnia dubia*, and the sphaerid clam, *Musculium transversum*. The MicrotoxTM assay measures the luminescence of *P. phosphoreum* (Bulich et al. 1981). Inhibition of this luminescence is considered a toxic response. The *S. capricornutum* assay measures the inhibition of photosynthetic activity of an algal culture as a measure of toxicity (Ross et al. 1988). The rotifer assay is a mortality test (Snell and Personne 1989). The *C. dubia* assay was the standard USEPA (1985) acute assay (48-hour mortality). The sphaerid or fingernail clam assay is based on measuring changes in filtering rates. The dilution water used in the toxicity tests and for maintaining the organisms was PerrierTM bottled water.

The fingernail clam filtering assay used in this study is based on observations by Aldridge et al. (1987), Sparks and Sandusky (1983), Sparks et al. (1981), and Anderson et al. (1978) that stresses, including toxicants, impair the ability of bivalves to filter particles from water (including the food particles on which the clams feed). The assay is outlined below and a detailed description is given in Sparks et al. (1992). Filtering rates are determined by measuring the fingernail clams' ability to filter yeast from a suspension of known concentration. Fingernail clams are first exposed to the porewater sample for one hour. They are then placed in a yeast suspension and allowed to filter for one hour. Two controls are used: the first consists of the yeast suspension alone and is used to determine the change in concentration due to settling of the yeast. The second control determines the baseline filtering rate of clams exposed for 1 hour in clean, uncontaminated water. The yeast concentrations are measured at the beginning and end of the filtering period. The filtering rates of the exposure and control tests are then determined by taking the initial yeast concentration minus the final concentration minus the amount settled divided by the weight of the test organisms. Filtering rates are expressed as the concentration of yeast filtered per unit weight of organism per unit time.

C_i = initial concentration of yeast

C_f = final concentration of yeast

W = wet weight of clams, in g (grams)

C_s = change in yeast concentration due to settling

$$\frac{C_i - C_f - C_s}{W} = \text{filtering rate in mg (milligrams) yeast/g clam/hour}$$

The exposure filtering rate is then compared to the control. The test result is a sublethal response (percent reduction in filtering rate, relative to the control) as opposed to an "all or none" (death or survival, toxic or nontoxic) type of response. The inhibition of the filtering performance of the clams is proportional to the severity of the stress (Sparks et al. 1992). For purposes of evaluating sediments for toxicity, it is useful to be able to rank sites based on relative toxicity. Only the 1990-1991 porewater samples were evaluated using this assay because it was not fully developed until late 1990.

The results of the various assays were standardized for easier comparisons. The treatment results were divided by the control results and then 1 was subtracted from the quotient. A negative value indicates inhibition (toxicity), a positive value indicates stimulation, and 0 indicates no response (no difference with respect to the control). If we use the fingernail clam filtering bioassay as an example:

T = test response to sample of sediment porewater

C = control response to uncontaminated dilution water

T = 3.4 mg yeast/g clam/hour

C = 6.5 mg yeast/g clam/hour

$T/C = 3.4/6.5 = .52$

$.52 - 1.00 = -.48$ A decline of 48% from the control value, a marked inhibition of the filtering ability of the clams.

Results of the *C. dubia* bioassay are expressed in toxicity units, as well as 48-hour LC50s, where toxicity units = $100/(48\text{-hour LC50})$. The 48-hour LC50 is the percent dilution of porewater (or treated porewater) that kills 50% of the test organisms in 48 hours. For example,

if a 7% solution (by volume) of porewater in dilution water is the LC50 (see site CS307.4, Table 4.1),

$$7\% = 48\text{-hour LC50}$$

$$100/\text{LC50} = 100/7 = 14.3 \text{ toxic units}$$

meaning that the toxicity in the porewater is more than 14 times the lethal dose.

3.5 Toxicity Identification and Evaluation Procedures

Samples exhibiting acute toxicity to *C. dubia* were subjected to Toxicity Identification and Evaluation (TIE) procedures developed at the USEPA's National Effluent Toxicity Assessment Center (NETAC). The goal is to separate toxicants from nontoxic compounds, using sample fractionation techniques in combination with bioassays to determine which fractions contain most of the toxicity. We used *C. dubia* as the TIE test organism, because it is a widely-accepted reference species. The TIE approach consists of three phases outlined in Figure 1.3.

3.5.1 Phase I characterizes the physical and chemical properties of the sample toxicants by altering or rendering biologically unavailable generic classes of compounds (Mount and Anderson-Carnahan 1988). After Phase I the toxicants are classified as having characteristics of cationic metals, non-polar organics, volatiles, oxidants, or substances not affected by Phase I methods. Phase I manipulations are outlined in Figure 1.4. The primary tool of Phase I is manipulation of sample pH. The questions asked are: (1) Is toxicity different at different pHs? (2) Does sample manipulation at different pHs affect toxicity? (3) Is toxicity attributable to cationic metals, such as copper or lead? (4) Is toxicity associated with oxidizing agents, such as chlorine or

chloramines? The graduated pH test answers the first question and is designed to indicate a pH-dependent toxicant such as un-ionized ammonia. The second question is answered by performing the following tests at different pHs: aeration, filtration and reverse-phase solid phase extraction (SPE). Aeration tests determine whether toxicity is attributable to volatile or oxidizable compounds. The filtration tests indicate whether toxicity is associated with filterable components. Reverse-phase SPE indicates whether toxicity is attributable to non-polar compounds. Presence of toxic cationic metals is indicated if addition of a chelating agent, ethylenediaminetetraacetic acid (EDTA), diminishes toxicity. Presence of chlorine or other oxidizing agents is indicated by a reduction in toxicity following addition of the reducing agent, sodium thiosulfate.

3.5.2 Phase II uses chemical fractionation techniques in parallel with toxicity tests to isolate suspected toxicants (Mount and Anderson-Carnahan 1989). Our Phase I results strongly implicated ammonia as a toxicant, so we retested the samples after selectively removing ammonia using a zeolite ion exchange resin, following the methods of Mount and Anderson-Carnahan (1989), Ankley et al. (1990), and Schubauer-Berigan and Ankley (1991). Zeolites are naturally-occurring or synthetically-created crystalline hydrated alkali-aluminum silicates. A column was prepared by packing a glass tube with a commercially available zeolite product. The sample was passed over the zeolite column using a metering pump, at a flow rate of approximately 10 ml/min (milliliters per minute). Post column samples were analyzed for total ammonia and screened for acute toxicity.

In addition to implicating ammonia, Phase I testing also indicated that toxicity in some samples was associated with non-polar organic materials and with material that was retained on the filters. We applied the following Phase II isolation techniques that were used in a similar situation by Schubauer-Berigan and Ankley (1991). To verify that toxicity was due in part to material retained on the filters, the filters were extracted with methylene chloride. Filters used in Phase I for samples from the Des Plaines River site DP277.0 and the Calumet Sag Channel site CS307.4 were soaked in 10 ml of methylene chloride for 1 hour. The solvent was evaporated from the beakers and dilution water was added to the same volume as the original filtered sample. The extracts then were screened for acute toxicity.

Having checked the toxicity of the material on the filters, we next investigated the nonpolar organics using solid phase C_{18} absorption columns and subsequent chromatography. To maximize the extraction of possible toxicants, filtration was omitted and porewater was centrifuged at 10,000 g for 30 minutes to remove particles that would clog the C_{18} column. The supernatant from the centrifugation step was checked for toxicity. If toxicity was present, a 200-ml sample of the supernatant was passed over a 6-ml C_{18} column that had been conditioned with 25 ml of methanol followed by 25 ml of MilliporeTM ultrapure water. Post column aliquots were collected after passage of 25 ml and 100 ml of methanol and tested for toxicity.

Toxicity was not recovered from the DP277.0 sample using 100% methanol elutions of the C_{18} columns as suggested by Mount and Anderson-Carnahan (1989), so we eluted the columns with increasingly nonpolar mixtures of methylene chloride in methanol (1, 5, 10, 15, 20, 25, 30, 35, 40, 45, 50, and 100%) as suggested by Schubauer-Berigan and Ankley

(1991). The elutriates were collected in hexane-rinsed scintillation vials. Toxicity may not have been recovered by methanol extractions because either the toxic compounds or the oil and grease they are sorbed to are highly nonpolar. The methylene chloride was evaporated and the sample restored to volume using methanol. The fractions were tested for toxicity using 150 μ l (microliters), 75 μ l, and 37.5 μ l of the fraction in 10 ml of dilution water. The methanol concentrations were below the 48-hour LC50 for *C. dubia*, so toxicity was attributed to the nonpolar organic solutes, rather than to the methanol solvent.

The toxic fractions were sent to Daily Analytical Laboratories in Peoria, Illinois for analysis on a Hewlett-Packard 5890A gas chromatograph with a 5970A Series mass selective detector along with a 7673A autosampler. The methanol concentrate was injected into a 30-m (meter) x 0.25-mm (millimeter)-i.d. DB-5 J&W capillary column. The temperature program was 40°C for 4 minutes followed by an increase at a rate of 10° C per minute to a peak of 300° C for 10 minutes. Run time was 40 minutes with a scan start time at 3 minutes. The peak detection threshold was 10,000 counts, with a threshold at 100 counts. A splitless injection mode was used along with a linear scanning method from 40-460 mhz (megahertz). The samples had 40 μ g (micrograms)/ml of internal standards of the following compounds; 1,4-Dichlorobenzene-d4, Naphthalene-d8, Acenaphthene-d10, Phenanthrene-d10, Chrysene-d12 and Perylene-d12. After the sample was analyzed by the mass selective detector, they were compared to library searches using the NIH (National Institutes of Health) EPA (U.S. Environmental Protection Agency) Mass Spectral Database. Identifications were based on the best fit with a minimum search fit of 70%.

3.5.3 Phase III confirms the identity of toxicants that are provisionally identified in Phases I and II. We employed two methods from the suite of Phase III techniques suggested by Mount (1988): (1) We correlated toxicity with measured concentrations of suspect chemicals in our test solutions, and (2) we compared the relative sensitivity of our test species to known toxicants and to our samples. The correlation analysis was performed on the toxicity tests which used the standard reference animal, *C. dubia*. As mentioned earlier, the correlation analyses used total ammonia concentrations, rather than un-ionized ammonia concentrations. The drift in pH during the toxicity tests made it impossible to calculate un-ionized ammonia concentrations based on measurements of total ammonia and the pH of the test solutions.

4.0 RESULTS

4.1 Relative Toxicity

There were marked differences in the responses of the five test organisms to sediment porewater from the same sites (Figure 4.1). Luminescence of the marine bacterium, *Photobacterium phosphoreum*, (Microtox test) was inhibited by 34% at SS313.0 on the Sanitary and Ship Canal and 32% at CS307.4 on the Calumet Sag Channel. Maximum stimulation of approximately 50% occurred at the next site upstream on the Calumet Sag Channel, CS318.5. Responses to porewaters from other sites were slight and variable, sometimes mildly inhibitory and sometimes mildly stimulatory.

Photosynthesis by the freshwater alga, *Selenastrum capricornutum*, was markedly stimulated, by a factor of nearly 2, by sediment porewaters from the mouth of Swan Lake, IR6.0, and the Sanitary and Ship Canal, SS310.0. Stimulation is an indication of nutrient enrichment; e.g., by nitrogen and phosphorus (Ross et al. 1988). The greatest inhibition, -86%, was caused by sediment porewater from Lake Chautauqua, IR125.5, although inhibition also occurred at IR72.0, IR281.1, SS313.0, SS315.3, and CS307.4.

A large percentage of the rotifers, *Branchionus calciflorus*, died in porewaters from Meredosia Lake (IR72) and Lake Chautauqua (IR125.5), but the rotifers exhibited no significant responses to samples taken anywhere else (Figure 4.1).

In contrast to the microorganisms (bacterium, alga, and rotifer), the macroinvertebrates *C. dubia* and *M. transversum* were remarkably

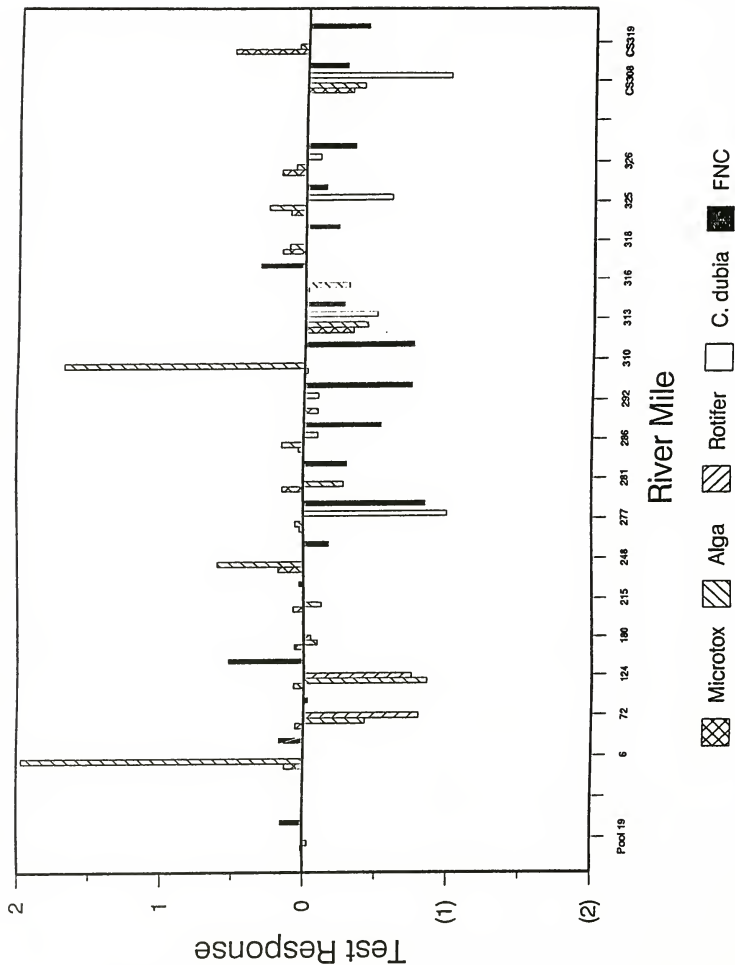


Figure 4.1. Responses of five organisms to porewaters from sediments of the Illinois Waterway and from a reference site (Pool 19 of the Upper Mississippi River). Numbers above zero represent stimulation, numbers below zero represent inhibition (toxicity). CS = Calumet Sag Channel. FNC = fingernail clams.

consistent in their responses to the sediment porewaters. Both organisms exhibited no inhibitory response to porewaters from the lower Illinois River or from the reference site in the Upper Mississippi River (Figure 4.1). The stimulation of filtering performance in the fingernail clam, *M. transversum*, may have been caused by favorable ratios of dissolved sodium, potassium, calcium, and magnesium salts in porewaters from the lower river. Anderson, Sparks and Paparo (1978) demonstrated the importance of these salts in regulating the beating of the cilia on the gills of the clams. Salts that affect the cilia are likely to affect filtering performance because the lateral cilia produce the water currents that bring food into the clam and the latero-frontal cilia act as filters. Also, the presence of organic matter in the sediment porewaters may have stimulated a feeding response in the clams, which are deposit feeders, as well as water column filterers. The clam and the water flea likewise are consistent in indicating toxicity in the upper waterway. Filtering performance in the clam was inhibited starting with sediment porewaters from IR248.2 near Marseilles and water flea mortality started at DP277.0, just above the mouth of the Du Page River near the Interstate 55 bridge. Sediment porewaters from 7 of the 13 upstream sites were toxic to *C. dubia*, and 12 of 13 inhibited the fingernail clam (Figure 4.1).

Since the fingernail clam is the organism of main interest in this study, the response of the clams is extracted from Figure 4.1 and presented separately in Figure 4.2. With the exception of a stimulatory response to porewater from one station in the Sanitary and Ship Canal, SS315.3, all the upper waterway stations exhibited some degree of

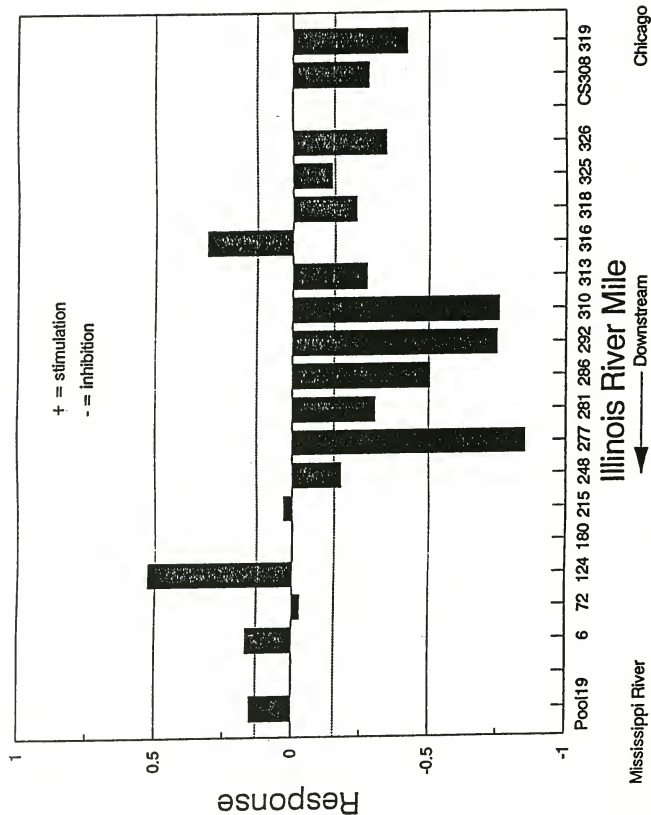


Figure 4.2. Responses of fingernail clams to porewaters from sediments of the Illinois Waterway and from a reference site (Pool 19 of the Upper Mississippi River). Numbers above zero represent stimulation, numbers below zero represent inhibition (toxicity). CS = Calumet Sag Channel.

toxicity to the fingernail clam, with the most toxic stations located in the reach between the mouth of the Du Page River, DP277.0, and the Summit-Stickney area, SS313.0.

4.2 Toxicity Identification Evaluation - Phase I

Standard toxicity identification evaluations (TIE) use *C. dubia* to determine whether various treatments reduce the toxicity of porewater. We felt it was unnecessary to use the nonstandard clam bioassay in TIE because the fingernail clams and *C. Dubia* responded similarly to the sediment porewaters, and *C. dubia* appeared to be an adequate surrogate for the clam.

Seven sites were acutely toxic to *C. dubia* in 1990 and 1991, with six sites in common between the two years (Table 4.1). One site on the upper Calumet Sag Channel, CS318.5, was acutely toxic in 1990 but not in 1991. The mouth of the Du Page River, DP277.0, was not sampled in 1990. The level of toxicity ranged from 1.1 to 14.3 times the lethal dose, with the greatest toxicity observed in the Calumet Sag Channel (CS307.4) in 1991. The second greatest toxicity (7 times the lethal dose) was also observed in 1991 near the mouth of the Du Page River (DP277.0). The following discussion of the TIE Phase I results is summarized by year, 1990 and 1991.

4.2.1 1990. The only sample manipulation that consistently reduced toxicity in the 1990 samples of sediment porewater was the graduated pH test (Table 4.2). Toxicity at pH 8.5 was greater than at pH 7.5 and pH 6.5 indicating a pH-dependent toxicant. Some ionic compounds, e.g., cationic metals, can be more toxic at a higher pH; however, EDTA chelation tests did not remove toxicity. Another common

Table 4.1. Toxicity of porewater to *Ceriodaphnia dubia* (48-hour LC50, reported as a % of porewater in test solution, and as toxic units, 100/LC50).

Site	1990		1991	
	LC50	Toxic Units	LC50	Toxic Units
CR 326.4	66 (48-79)	1.5	66 (48-79)	1.5
CR 324.8	71	1.4	89	1.1
CS 318.5	67 (44-91)	1.5	>100	----
CS 307.4	62 (47-80)	1.6	7 (5-12)	14.3
SS 317.0	>100	----	>100	----
SS 315.3	NS	----	>100	----
SS 313.0	51 (25-80)	2.0	95	1.1
SS 310.0	NS	----	>100	----
SS 292.2	71	1.4	35	2.8
DP 286.3	71	1.4	71	1.4
DP 281.1	NS	----	>100	----
DP 277.0	NS	----	14 (10-23)	7.1
IR 248.2	>100	----	>100	----
IR 215.0	>100	----	>100	----
IR 180.0	>100	----	>100	----
IR 125.5	>100	----	>100	----
IR 72.0	>100	----	>100	----
IR 6.0	>100	----	>100	----

Notes:

a. >100 indicates that 100% porewater did not kill at least half the test organisms in 48 hours.

b. Dashes (----) indicate that toxic units could not be calculated because porewater was not lethal within the 48-hour exposure period.

c. Numbers in parentheses indicate range of dilutions that caused 50% mortality in 48 hours (48-hour LC50s).

NS=not sampled

Table 4.2. Results of treating sediment porewater to reduce toxicity and characterize the toxicant. Porewater was obtained from acutely toxic Illinois Waterway sediments in 1990.

Phase I Treatments	DP 286.3	Sample Site		
		SS 292.2	SS 313.0	CS 307.4
pH adjustment	NR	NR	NR	NR
Aeration	NR	NR	NR	NR
Filtration	NR	NR	NR	NR
Reverse-phase SPE	NR	NR	NR	NR
Oxidation reduction	NR	NR	NR	NR
EDTA chelation	NR	NR	NR	NR
Graduated pH	R	R	R	R

Phase I Treatments	CS 318.5	Sample Site	
		CR 324.8	CR 326.4
pH adjustment	NR	NR	NR
Aeration	NR	NR	NR
Filtration	NR	NR	NR
Reverse-phase SPE	NR	NR	NR
Oxidation reduction	NR	NR	NR
EDTA chelation	NR	NR	NR
Graduated pH	R	R	R

NR= No reduction in toxicity
 R= Reduction in toxicity
 SPE= solid phase extraction

aquatic toxicant that is strongly pH-dependent is ammonia. Total ammonia concentrations in the acutely toxic samples ranged from 32.7 mg/l (milligrams per liter) to 59.8 mg/l (Table 4.3).

4.2.2 1991. Five of the seven sites evaluated in 1991 had the same characterization pattern as in 1990 (Table 4.4). The only manipulation to consistently reduce toxicity was the graduated pH test, again indicating a pH-dependent toxicant such as ammonia (Table 4.3). Total ammonia concentrations in the 1991 samples ranged from 28.6 mg/l to 51.2 mg/l (Table 4.3). The characterization pattern differed for porewaters from DP277.0 on the Des Plaines River and CS307.4 on the Calumet Sag Channel (Table 4.4). Toxicity in these porewaters was reduced by filtration and solid phase extraction with a C₁₈ column, indicating that toxicity is due to non-polar organic compounds associated with filterable particles. These samples contained visible quantities of oil.

In summary, Phase I results from 1990 and 1991 indicate that acute toxicity in most sediment porewaters from the Upper Illinois Waterway is attributable to a pH-dependent toxicant, most likely ammonia. Porewater from one location in the lower Des Plaines River and one location in the lower Calumet Sag Channel contained toxicity attributable to non-polar organics associated with oil or grease.

4.3 Toxicity Identification Evaluation - Phase II

Phase II techniques were used to isolate toxicants in porewaters from the seven sites where ammonia was suspect and the two sites where non-polar organics were suspect. The zeolite columns completely removed acute toxicity from porewaters where ammonia was suspect (Table 4.5).

Table 4.3. Toxicity and ammonia concentrations in sediment porewater in 1990 and 1991. Toxic units = $100/LC50$, where $LC50$ is the % dilution that kills 50% of the exposed *Ceriodaphnia dubia* in 48 hours. Ammonia concentrations in the porewater are expressed as total ammonia nitrogen, N, in mg/l.

1990		
<u>Site</u>	<u>Toxic Units</u>	<u>Total Ammonia-N mg/l</u>
CR 324.8	1.4	37.8
CR 326.4	1.5	25.6
CS 307.4	1.6	35.4
CS 318.5	1.5	42.7
SS 292.2	1.4	41.5
SS 313.0	2.0	59.8
DP 286.3	1.4	23.8
1991		
CR 324.8	1.1	34.2
CR 326.4	1.5	51.2
SS 292.2	2.8	33.5
SS 313.0	1.1	28.6
DP 286.3	1.4	30.5

Table 4.4. Results of treating sediment porewater to reduce toxicity and characterize the toxicant. Porewater was obtained from acutely toxic Illinois Waterway sediments in 1991.

Phase I Treatments	Sample Site			
	DP 277.0	DP 286.3	SS 292.2	CS 307.4
pH adjustment	NR	NR	NR	NR
Aeration	NR	NR	NR	NR
Filtration	R	NR	NR	R
Reverse-phase SPE	R	NR	NR	R
Oxidation reduction	NR	NR	NR	NR
EDTA chelation	NR	NR	NR	NR
Graduated pH	NR	R	R	NR

Phase I Treatments	Sample Site		
	CS 313.0	CR 324.6	CR 326.4
pH adjustment	NR	NR	NR
Aeration	NR	NR	NR
Filtration	NR	NR	NR
Reverse-phase SPE	NR	NR	NR
Oxidation reduction	NR	NR	NR
EDTA chelation	NR	NR	NR
Graduated pH	R	R	R

NR= No reduction in toxicity

R= Reduction in toxicity

SPE= solid phase extraction

Table 4.5. Results of treating sediment porewater with zeolite to remove ammonia. Porewater was obtained from acutely toxic Illinois Waterway sediments.

Site	1990			
	Pre-Zeolite		Post Zeolite	
	Ammonia-N (mg/l)	Toxicity	Ammonia-N (mg/l)	Toxicity
CR 326.4	25.62	T	1.70	NT
CR 324.8	37.82	T	1.46	NT
CS 318.5	42.70	T	1.98	NT
CS 307.4	35.38	T	2.24	NT
SS 313.0	59.78	T	2.58	NT
SS 292.2	41.48	T	1.22	NT
DP 286.3	23.67	T	4.88	NT
	1991			
CR 326.4	51.29	T	1.86	NT
CR 324.8	34.16	T	1.70	NT
SS 313.0	28.60	T	3.05	NT
SS 292.2	33.55	T	1.95	NT
DP 286.3	30.50	T	1.70	NT

T = Acute toxicity was present, as determined by toxicity tests with *Ceriodaphnia dubia*.
 NT = No acute toxicity

Since the zeolite selectively removes ammonia, these results support identification of ammonia as the toxicant.

The suspect nonpolar organics at sites CS307.4 and DP281.1 seemed to have different chemical and physical properties because no toxicity could be obtained from DP281.1 by column absorption and elution with methanol alone, whereas CS307.4 did yield toxicity with the methanol extraction (Table 4.6). Moreover, toxic materials were eluted by a wider range of methylene chloride/methanol mixtures (20-50%) from the CS307.4 sample than from the DP277.0 sample (25-40%, Table 4.7). Also, the greatest toxicity in porewater sample DP281.1 was associated with residue left on the filters after passage of porewater, whereas the greatest toxicity in sample CS307.4 was in supernatant left after centrifuging out most of the particles (Table 4.6). The DP277.0 elutriate contained no organics detectable by gc-mass spectrography, whereas 34 organic compounds were detected in the CS307.4 elutriate (Table 4.8). This was surprising because the DP277.0 elutriates contained toxicity (Table 4.7), but perhaps there were undetectable quantities of nonpolar organics that were highly toxic.

The elutriates from sample CS307.4 contained different combinations of nonpolar organics (Table 4.8). No compounds were found above the detection limits in the 20% fraction. The 25% fraction contained the polycyclic aromatic hydrocarbon (PAH) naphthalene. The 30% fraction contained primarily cyclic and branched hydrocarbons (cyclohexane, octane) and PAHs. The 35-50% fractions contained numerous long chain hydrocarbons such as heptadecane, undecane and dodecane. The 40 and 45% fractions also contained the alkenes, eicosene and dotriacontanol. In general, toxicity in these samples appears to be primarily due to petroleum hydrocarbons and PAHs. Scubauer-Berigan and Ankley (1991)

Table 4.6. Toxicity of sediment porewater following fractionation by filtration, centrifugation, and column absorption and extraction. Fractions were tested for toxicity using *Ceriodaphnia dubia*. Toxicity is expressed as the 48-hour LC50, reported as % of sample fraction in test solution, and as toxic units, (100/LC50). Numbers in parentheses indicate a range of dilutions that caused 50% mortality in 48 hours.

	DP 277.0		CS 307.4	
	LC50	Toxic Units	LC50	Toxic Units
^a Filter extraction	27 (18-40)	3.7	62 (48-71)	1.6
^b Centrifugation whole sample	71	1.4	9	11.1
^c Post C ₁₈ (25 ml)	>100	----	18	5.6
^c Post C ₁₈ (100 ml)	>100	----	18	5.6

^aPorewater was filtered, then the filters were extracted with methylene chloride. Extracts tested for toxicity.

^bCentrifugation at 10,000 g for 30 minutes to settle the particles in the porewater. Supernatant tested for toxicity.

^c200-ml samples of supernatants from b were passed through absorption columns, then aliquots were taken after passage of 25 ml and 100 ml of methanol through the columns. Aliquots tested for toxicity.

>100 indicates that the undiluted fraction did not kill at least half the test organisms in 48 hours.

----indicates that toxic units could not be calculated because the undiluted fraction was not lethal within the 48-hour exposure period.

Table 4.7 Toxicity of extracts from sediment porewater. The porewater was obtained from sediments at site CS 307.4 and site DP 277.0 where nonpolar organic chemicals were suspected of contributing to toxicity. The porewater was passed through C₁₈ absorption columns and then the columns were eluted with increasingly nonpolar mixtures of methylene chloride in methanol (1%-100% methylene chloride). The elutriates were tested for toxicity on *Ceriodaphnia dubia*.

Fraction of Methylene Chloride in Methanol	Site DP 277.0	Site CS 307.4
100%	NT	NT
50%	NT	T
45%	NT	T
40%	T	T
35%	T	T
30%	T	T
25%	T	T
20%	NT	T
15%	NT	NT
10%	NT	NT
5%	NT	NT
1%	NT	NT

T = Toxic
NT = Not Toxic

Table 4.8 Constituents of elutriates from solid phase C₁₈ absorption columns. Toxic sediment porewaters from sites CS 307.4 and DP 277.0 were passed through the columns, which then were elutriated with mixtures of methylene chloride in methanol. The elutriates were analyzed with a gas chromatograph. Values in table are concentrations, in mg/l, calculated from areas under the peaks in the chromatographs.

Chemical	CS 307.4					DP 277.0
	Methylene 30	chloride 35	/methanol 40	% 45	 50	
cyclohexadecane	--	--	--	--	11	No peaks above detection limits
cyclohexane, dimethyl	38	--	--	--	--	
cyclohexane, trimethyl	--	--	--	--	48	
cyclohexane, methylpropyl	--	--	--	--	32	
cyclopentane, 1-methyl- 3-4-(1-methylethyl)	--	--	--	61	--	
cyclopentane, methyl propenyl	--	--	26	--	--	
decane, trimethyl	--	--	--	12	--	
dodecane, trimethyl	--	10	54	50	18	
1-dotriacontanol	--	--	22	--	--	
3-eicosene	--	--	21	32	--	
5-eicosene	--	--	--	13	--	
heneicosan, ethylpropyl	--	--	--	18	--	
heptacosane	--	--	--	--	44	
heptadecane	--	--	--	--	16	
heptadecane, trimethyl	--	--	--	76	--	
heptadecane, tetramethyl	--	18	44	49	46	
heptane, 3-ethyl-5-methyl	--	15	--	38	--	
1-heptanol, 2-propyl	--	13	--	--	--	
nonadecane	--	--	21	--	--	
nonahexacontanoic acid	--	--	--	--	12	
nonane, dimethyl	--	--	16	--	--	
octane, trimethyl	--	--	--	29	--	
octane, dimethyl	10	--	--	--	--	
octadecane, chloro	--	--	--	17	--	
3-octadecanol	--	--	--	13	--	
naphthalene, decahydro-2-methyl	10	28	--	--	--	
tetratetracontane	--	--	--	36	48	
tritetracontane	--	--	22	--	11	
tridecane, methyl	--	--	14	13	--	
tritetracontane	--	--	--	16	--	
undecane, dimethyl	--	--	--	19	--	
undecane, 2,5-dimethyl	--	23	--	--	--	
undecane, 3,6-dimethyl	--	--	36	--	--	
undecane, 6-methyl	--	21	--	--	--	

-- = not detected

identified non-polar organics associated with oil and grease as a source of toxicity in sediments in the upstream portions of the Calumet Sag Channel and Lake Calumet.

4.4 Toxicity Identification Evaluation - Phase III

Toxicity in sediment porewaters from the upper Illinois Waterway is correlated with total ammonia concentrations ($r = 0.85$, Figure 4.3). Jones and Lee (1988) found that of more than 30 contaminants measured in sediments from New York Harbor, only ammonia concentrations correlated to observed toxicity in grass shrimp. Ankley et al. (1990) identified ammonia as a major toxicant in sediments from the lower Fox River and Green Bay, Wisconsin.

The fingernail clam, *Musculium transversum*, is sensitive to ammonia. Anderson, Sparks and Paparo (1978) found that un-ionized ammonia concentrations of 0.08-0.09 mg/l inhibited the cilia on the gills of the clams, and the growth of the clams in the laboratory was reduced at concentrations between 0.20 and 0.34 mg/l $\text{NH}_3\text{-N}$. Un-ionized ammonia concentrations greater than these are likely to occur in sediment porewaters of the upper Illinois Waterway, based on total ammonia concentrations we measured (23.8-59.8 mg/l $\text{NH}_3\text{-N}$) and pH ranges known to occur in the water column. The clams must draw oxygenated water from the water column down their burrows to survive, and in doing so, they might shift the pH from the low levels characteristic of anaerobic sediments to higher levels characteristic of the water column, thereby increasing the fraction of the total ammonia that exists in the toxic un-ionized form.

In summary, several lines of evidence lead to the conclusion that ammonia in sediment porewater was limiting macroinvertebrate populations in the Illinois Waterway at the time this study was conducted. First,

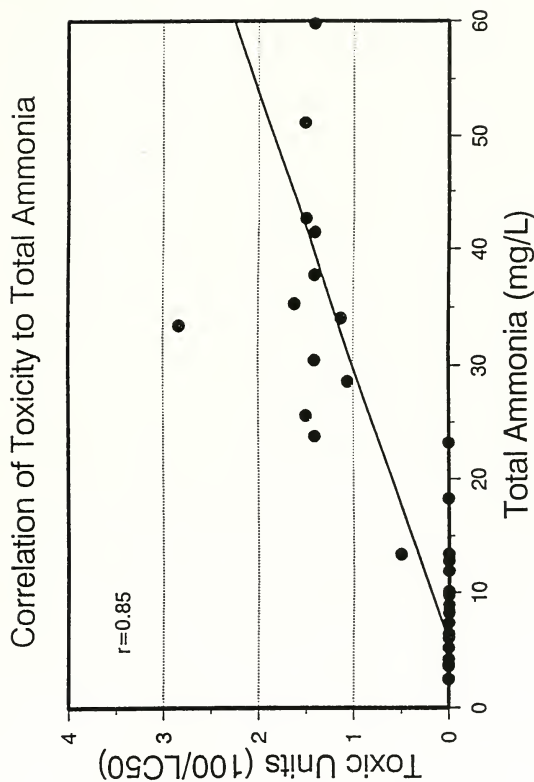


Figure 4.3 Correlation of toxicity with total ammonia concentrations (mg/L ammonia as nitrogen) in sediment porewaters. Toxicity is expressed in toxic units, 100/LC50, where LC50 is the dilution of porewater in clean water that causes half of the Ceriodaphnia dubia to die within 48 hours.

the porewater contains a pH-sensitive toxicant that is not affected by chelation with EDTA, as heavy metals would be (Tables 4.2 and 4.4). Second, toxicity to both *C. dubia* and the fingernail clam *M. transversum* is associated with total ammonia concentrations in sediment porewater (Table 4.3, and Figures 4.1, 4.2, and 4.3). Third, removal of ammonia by treatment with zeolite removes the toxicity (Table 4.5). Finally, *M. transversum* is known to be sensitive to un-ionized ammonia at levels that are likely to occur in the porewaters.

Not all the toxicity found in the upper Illinois Waterway was associated with ammonia. Porewaters from the Calumet Sag Channel (CS307.4) and the lower Des Plaines River (DP277.0) contained visible signs of oil and grease and toxicity associated with PAHs (including naphthalene) and other compounds found in petroleum.

4.5 Sensitivity of Recolonizing Clams

Much to our surprise, we found several species of fingernail clams, including *Musculium transversum*, at several sites in the upper Illinois Waterway: the Chicago Sanitary and Ship Canal (SS317.0), the North Branch of the Chicago River (CR326.4), and the Calumet Sag Channel (CS318.5). Biologists from the Long Term Resource Monitoring (LTRM) Station at Havana also reported finding fingernail clams in mud they happened to bring up on their sampling nets and boat anchors.

We wondered if these clams had acquired some resistance to the toxicants in the sediments, so we tested their responses to a sediment sample from the Chicago Sanitary and Ship Canal (SS317.0) and another from the Calumet Sag Channel (CS318.5). At the same time, we tested clams from Swan Lake on the lower Illinois River, where we had obtained

all the clams used in our previous bioassays. This work was done outside the scope of our original research proposal and is of a very preliminary nature.

The preliminary results support the hypothesis of differential resistance. The clams from the Sanitary and Ship Canal showed virtually no impairment of filtering performance in response to porewater from the place where they were taken or to the Calumet Sag porewater (see below). The clams from Swan Lake were sensitive (36% decline in filtering performance) to porewater from the Sanitary and Ship Canal, while they had only a slight negative response, comparable to the Cal-Sag clams, to the Cal-Sag porewater. The Ship Canal porewater was not tested on the Cal-Sag clams.

<u>SOURCE OF CLAMS</u>	<u>SOURCE OF POREWATER</u>	
	<u>SS317.0</u>	<u>CS318.5</u>
CS318.5	-----	-0.17
SS317.0	-0.08	0.00
Swan Lake	-0.36	-0.13

5.0 DISCUSSION

Two different patterns of toxicity occur in the sediment porewaters of the Illinois Waterway. There is a gradient of increasing toxicity in the upstream direction, associated with increasing concentrations of total ammonia in the sediments. The second pattern is characterized by patches of toxicity associated with polycyclic aromatic hydrocarbons (PAHs), such as naphthalene, and long-chain hydrocarbons, both evidently derived from petroleum. One of the latter sites was located on the lower Des Plaines River section of the waterway, near several refineries. Previous studies have measured elevated levels of metals, pesticides, PAHs, and PCBs in the sediments of the upper Illinois Waterway (IEPA 1990) and demonstrated that sediments are toxic (Sparks et al. 1981; Blodgett et al. 1984; Schubauer-Berigan and Ankley 1991). The two toxicity problems might even be related: Ankley et al. (1991) suggested that natural microbial processes in aquatic ecosystems may be compromised by organic loading or selective toxicity. The alteration of microbial processes could play a role in the incidence of ammonia accumulation and subsequent toxicity in sediments in the Upper Illinois Waterway.

It is well established that certain sediments can contain high concentrations of ammonia (Keeney 1973, Berner 1980). Nitrogen-containing organic matter is decomposed in sediments by heterotrophic bacteria. The amount of ammonification that takes place depends in part on oxygen availability (Kleerekoper 1953). Ammonia can accumulate to toxic levels under anaerobic conditions (Berner 1980). Serruya (1974) found that ammonia formation is greatest about 10 cm (centimeters) below the sediment-water interface. In this situation, ammonia probably diffuses

from the deeper sediments to surficial sediments, and perhaps even to the overlying water, especially if sediments are resuspended by currents or boat- or wind-driven waves. The fingernail clam, *Musculium transversum*, the organism of primary interest in this study, makes shallow burrows in the sediment and may be exposed to much higher levels of ammonia than organisms living in the water column, at the mud-water interface, or on plants, rocks, and woody debris.

Ammonia toxicity is due to the un-ionized (NH_3) form (USEPA 1985). The proportion of total ammonia existing in the un-ionized form is controlled primarily by pH and temperature (Emerson et al. 1975). The pH of sediments can fluctuate dramatically on a seasonal basis, and the pH of the overlying water can fluctuate daily, so that episodes of toxicity may occur even if the total ammonia concentration remains relatively constant. Ammonia loading of rivers tends to increase during winter because the microorganism-mediated conversion of ammonia to nitrate stops at cold temperatures. Also, aquatic vegetation does not remove ammonia (a plant nutrient) during winter dormancy. Water quality standards frequently allow higher levels of ammonia in the winter because the proportion of total ammonia existing in the toxic, un-ionized form is less at cold temperatures. However, the sensitivity of fish to ammonia increases at cold temperatures, so even though there may be less un-ionized ammonia, acute toxicity may still occur (Reinbold and Pescitelli 1990). Research is needed to determine the effect of cold temperatures on the sensitivity of invertebrates, as well as fish, to ammonia.

Musculium transversum is sensitive to ammonia. Anderson, Sparks and Paparo (1978) found that un-ionized ammonia concentrations of 0.08-0.09 mg/l (expressed as un-ionized ammonia nitrogen, $\text{NH}_3\text{-N}$, in mg/l)

inhibited the cilia on the gills of the clams, and the growth of the clams in the laboratory was reduced at concentrations between 0.20 and 0.34 mg/l $\text{NH}_3\text{-N}$. The *C. dubia* acute LC50 for ammonia is 1.04 mg/l $\text{NH}_3\text{-N}$ (Ankley et al. 1990). Arthur et al. (1987) reported un-ionized ammonia toxicity to 5 invertebrates ranged from 1.95 to 18.3 mg/l $\text{NH}_3\text{-N}$ and mollusks (snails) were most sensitive.

SPECIES	LC50 (mg/l)
Snail	
<i>Physa gyrina</i> - adult	1.95
<i>Helisoma trivolvis</i> - adult	2.17
Amphipod	
<i>Crangonyx pseudogracilis</i> - adult	3.12
Mayfly	
<i>Callibaetis skokianus</i> - nymph	3.12
Isopod	
<i>Asellus racovitzai</i> - adult	5.02
Caddisfly	
<i>Philarctus giaeris</i> - larvae	10.1
Crayfish	
<i>Orconectes immunis</i> - adult	18.3

Concentrations of this magnitude (1.0-8.0 mg/l $\text{NH}_3\text{-N}$) are commonly found in the sediments in the Upper Illinois Waterway, based on total ammonia concentrations (23.8-59.8 mg/l) and naturally occurring pHs. Ammonia places organisms in double jeopardy because it exerts an oxygen demand in the process of nitrification (conversion to nitrites and then nitrates) and low oxygen levels place organisms under additional stress (USEPA 1985). Ammonification may be occurring in the deep, anaerobic zones of the sediments and nitrification in the shallower, aerobic

zones, or in the boundary water at the sediment surface, so benthic invertebrates are exposed to the worst of both worlds. They are exposed to ammonia and to low oxygen at the same time.

The highest ammonia concentrations in sediments are associated with nitrogen-enriched sediments or high organic loading, as from sewage treatment plants (Brezonik 1973; Ankley et al. 1990; and Schubauer-Berigan and Ankley 1991). Although most sewage treatment plants remove a substantial portion of carbon in municipal waste, most do not remove nitrogen, but convert it from ammonia into nitrate. It is possible that nitrate is carried down into the sediments where it is converted back into ammonia in the anaerobic zones. If this is the case, ammonia toxicity in the sediments might be reduced by reducing the nitrogen loading of the river.

During the course of this study, several species of fingernail clams, including *M. transversum*, reappeared in the Chicago area waterways and in the Illinois River at Peoria and Havana. There are at least four possible explanations for this surprising reappearance of clams in the same general areas where the porewaters tested toxic. First, we found that clams recolonizing the upper Illinois were more resistant to ammonia than the clams from the lower Illinois, where the organisms were obtained for all of the early bioassays. Second, our previous research demonstrated that the surface layers of sediment in some areas were less toxic than layers a few centimeters deeper (Sparks, Sandusky and Paparo 1981; Blodgett et al. 1984). Toxicity may have been overestimated in tests where surface and deep layers of sediment were mixed prior to testing. Third, toxic episodes may be brief and infrequent, allowing organisms to colonize in between episodes. Fourth, the distribution of

toxicity in sediments may be extremely patchy, so that healthy organisms are found adjacent to barren areas. If the latter two hypotheses prove to be true, toxicity in the Illinois River has changed recently from a widespread, chronic problem to a more localized or episodic problem. Reduction of toxicity in surface sediments may reflect recent reductions in ammonia loading from sewage treatment plants in the Chicago area, although it is not clear whether the sources of ammonia in the porewaters are effluents, the deeper layers of sediments (as described above), or both.

We remind the reader that all the toxicity tests we conducted were short-term, acute tests. The fingernail clams, *Musculium transversum*, were exposed to sediment porewater for only 1 hour and then their filtering performance was tested in clean dilution water. The water flea, *Ceriodaphnia dubia*, was exposed to porewater for just 48 hours. The organisms in the waterways are exposed to contaminants for their entire life spans. In the past, more sensitive tests with fingernail clams have demonstrated toxicity even in downriver sediments, including Peoria Lake and Quiver Lake (Sparks, Sandusky and Paparo 1981).

In addition to being a problem for the benthic invertebrates that fish feed upon, ammonia in the Illinois Waterway may be a problem for the fish themselves. In 1987, the U.S. Fish and Wildlife Service simulated resuspension of bottom sediments by boat- or wind-driven waves by stirring sediments in clean water, allowing the sediment to settle for 24 to 48 hours, then exposing larval fathead minnows, *Pimephales promelas*, to the water. Water mixed with surface sediments from the Chicago River and the Des Plaines River killed all the fish within 24 hours. Surface sediments from Lake Chautauqua, a bottomland lake and federal

wildlife refuge along the Illinois River at Havana, killed 15% of the test fish in 96 hours; deeper sediments, taken at the 30.5-45.7 cm (12- to 18-inch) depth, killed 25%. Fish mortality correlated ($R = 0.71$, $P < 0.01$) with the concentration of un-ionized ammonia released from the sediment and both ammonia and fish mortality increased upstream toward Chicago. The Long Term Research Monitoring Station (LTRM) at Havana started measuring ammonia concentrations in Anderson Lake, a floodplain lake of the Illinois River and a state fish and wildlife area, on 1 May 1990, 2 days after a fish kill. The total ammonia nitrogen concentration was 0.90 mg/l and the un-ionized ammonia nitrogen was calculated to be 0.36 mg/l at the temperature of 16.6° C and pH of 9.34. $\text{NH}_3\text{-N}$ concentrations of 0.32 mg/l at 3-5° C and 1.35 mg/l at 24-25° C were acutely lethal to bluegill sunfish, *Lepomis macrochirus* (Reinbold and Pescitelli 1990). The fish kill might have been caused by ammonia, if the un-ionized ammonia had peaked at higher concentrations before our samples were taken.

Elevated un-ionized ammonia concentrations might be triggered by resuspension of sediments or episodes of elevated pH resulting from phytoplankton blooms. Plants remove carbon dioxide from the water, in the form of carbonic acid and bicarbonate, and thereby elevate the pH of the water, which in turn increases the proportion of ammonia existing in the toxic, un-ionized form. The Havana LTRM station (unpublished data) measured pHs as high as 10.12 in backwater lakes of the Illinois River in July 1990 and values between 9.0 and 10.0 occur fairly often. Episodes of acute ammonia toxicity thus may be occurring sporadically in places other than just the upper Illinois River, and it takes only one brief episode per year to kill or reduce populations of invertebrates or

fish that take many months or years to build up. Potential sources of ammonia or nitrogen, besides sewage plants and anaerobic sediments, include industrial plants (especially refineries and munitions plants), feedlots, and agricultural fields.

Although a general recovery does seem to be beginning in the Illinois River, with the return of fingernail clams in some areas where they have been absent at least 30 years and appearance of largemouth bass throughout the Illinois River proper, the pace and permanence of recovery still appears to be threatened by ammonia, even if the problem now turns out to be episodic instead of chronic. Reports of fingernail clam and mussel die-offs in the Upper Mississippi River and other rivers (Wilson et al. submitted; Blodgett and Sparks 1987; Neves 1987) indicate that drastic population declines in macroinvertebrates that burrow in sediments are not unique to the Illinois River.

6.0 LITERATURE CITED

- Adams, W.J., R.A. Kimerle, and R.G. Mosher. 1985. Aquatic safety assessment of chemicals sorbed to sediments. Pages 429-453 in R.D. Cardwell, R. Purdy, and R.C. Bahner, eds. Aquatic Toxicity and Hazard Assessment: Seventh Symposium, ASTM 854, American Society for Testing and Materials, Philadelphia, PA.
- Aldridge, D.W., B.S. Payne, and A.C. Miller. 1987. The effects of intermittent exposure to suspended solids and turbulence on three species of freshwater mussels. *Environmental Pollution* 45:17-28.
- Anderson, K.B. 1977. *Musculium transversum* in the Illinois River and an acute potassium bioassay method for the species. M.S. Thesis, Western Illinois University, Macomb, IL. 79 pp.
- Anderson, K.B., R.E. Sparks, and A.A. Paparo. 1978. Rapid assessment of water quality using the fingernail clam, *Musculium transversum*. University of Illinois Water Resources Center, Center Research Report No. 133.
- Anderson, R.V., D.M. Day, M. Demissie, F.S. Dillon, J.W. Grubaugh, M.S. Henebry, K.S. Lubinski, and R.E. Sparks. 1984. Flows, equations and input values for the nine state-variable biological model, Pool 19, Mississippi River--second generation. A computer-modelling project of the Large-River, Long-Term Ecological Research Project (LTER). Unpublished report. 106 pp.
- Ankley, G.T., A. Katko, and J. Arthur. 1990. Identification of ammonia as an important sediment-associated toxicant in the lower Fox River and Green Bay, Wisconsin. *Environmental Toxicology and Chemistry* 9:313-322.
- Ankley, G.T., K. Lodge, D.J. Call, M.D. Balcer, L.T. Brooke, P.M. Cook, R.G. Kreis, Jr., A.R. Carlson, R.D. Johnson, G.J. Niemi, R.A. Hoke, C.W. West, J. P. Giesy, P.D. Jones, and Z.C. Fuying. 1991. Integrated assessment of contaminated sediments in the lower Fox River and Green Bay, Wisconsin. *Ecotoxicology and Environmental Safety* 23:46-63.
- APHA. 1989. Standard methods for the examination of water and wastewater. 17th Edition, American Public Health Association, Washington, D.C.
- Arthur, J.W., W.W. Corlis, K.N. Allen, and S.F. Hedtke. 1987. Seasonal toxicity of ammonia to five fish and nine invertebrate species. *Bulletin of Environmental Contamination and Toxicology* 38:324-331.
- ASTM. 1982. Annual book of ASTM standards. Part 31. Water. American Society for Testing and Materials, Philadelphia, PA. 1554 pp.
- ASTM. 1991. Annual book of ASTM standards. Water. American Society for Testing and Materials, Philadelphia, PA.

- Bellrose, F.C., F.L. Paveglio, Jr., and D.W. Steffeck. 1979. Waterfowl populations and the changing environment of the Illinois River Valley. Illinois Natural History Survey Bulletin 32(1):1-54.
- Berner, R.A. 1980. Early Diagenesis: a theoretical approach. Princeton University Press, Princeton, NJ.
- Bhowmik, N.G., and M. Demissie. 1989. Sedimentation in the Illinois River valley and backwater lakes. Journal of Hydrology 105:187-195.
- Blodgett, K.D., R.E. Sparks, A.A. Paparo, R.A. Cahill, and R.V. Anderson. 1984. Distribution of toxicity in sediments of the Illinois Waterway. Pages 293-308 in W. Blake-Coleman, ed. Proceedings of the Conference of Urban Effects on Water Quality and Quantity. Urbana, IL 20-21 October 1983.
- Blodgett, K.D., and R.E. Sparks. 1987. Documentation of a mussel die-off in Pools 14 and 15 of the Upper Mississippi River. Pages 76-90 in R.J. Neves, ed. Proceedings of the workshop on Die-Offs of Freshwater Mussels in the United States. U.S. Fish and Wildlife Service and Upper Mississippi River Conservation Committee. 23-25 June, Davenport, IA.
- Brezonik, P.L. 1973. Nitrogen sources and cycling in natural waters U.S. Environmental Protection Agency, National Environmental Research Center, Washington, D.C. EPA600/3-73-002. 167 pp.
- Bulich, A.A., M.W. Greene, and D.L. Isenberg. 1981. Reliability of the bacterial luminescence assay for the determination of the toxicity of pure compounds and complex effluents. Pages 338-347 in ASTM-STP No. 737, Philadelphia.
- Burton, G.A. 1991. Assessing the toxicity of freshwater sediments. Environmental Toxicology and Chemistry 10(2):1585-1627.
- Cahill, R.A., and J.D. Steele. 1986. Inorganic composition and sedimentation rates of backwater lakes associated with the Illinois River. Illinois Department of Energy and Natural Resources, State Geological Survey Division, Environmental Geology Notes 115, Champaign. 61 pp.
- Cahill, R.A., and A.D. Autrey. 1987. Measurement of 210 Pb, 137 Cs, organic carbon and trace elements in sediments of the Illinois and Mississippi rivers. Journal of Radioanalytical and Nuclear Chemistry 110(1):197-205.
- Carlson, A.R., G.L. Phipps, V.R. Mattson, P.A. Kosian, and A.M. Cotter. 1991. The role of acid-volatile sulfide in determining cadmium bioavailability and toxicity in freshwater sediments. Environmental Toxicology and Chemistry 10(10):1309-1319.

- Carr, R.S., J.W. Williams, and C.T.B. Fragata. 1989. Development and evaluation of anovel marine sediment porewater toxicity test with the polychaete *Dinophilus gyrociliatus*. *Environmental Toxicology and Chemistry* 8(6):533-543.
- Coleman, J.A., and R.F. Sanzolone. 1991. Surface-water-quality assessments of the upper Illinois River basin in Illinois, Indiana, and Wisconsin: geochemical data for fine-fraction streambed sediment from high- and low-order streams, 1987. U.S. Geological Survey Open-File Report 90-571. 108 pp.
- Connell, D.W., M. Bowman, and D.W. Hawker. 1988. Bioaccumulation of chlorinated hydrocarbons from sediment by oligochaetes. *Ecotoxicology and Environmental Safety* 16:293-302.
- Di Toro, D.M., J.D. Mahony, D.J. Hansen, K.J. Scott, M.B. Hicks, S.M. Mays, and M.S. Redmond. 1990. Toxicity of cadmium in sediments: the role of acid volatile sulfide. *Environmental Toxicology and Chemistry* 9:1489-1504.
- Di Toro, D.M., C. Zarba, D.J. Hansen, W. Berry, R.C. Swartz, C.E. Cowan, S.P. Pavla, H.E. Allen, H.A. Homssand, P.R. Paquin. 1991. Technical basis for establishing sediment quality criteria for non-toxic chemicals using equilibrium partitioning. *Environmental Toxicology and Chemistry* 10(12):1541-1583.
- Di Toro, D.M., J.D. Mahony, D.J. Hansen, L.J. Scott, A.R. Carlson, and G.T. Ankley. 1992. Acid volatile sulfide predicts the acute toxicity of cadmium and nickel in sediments. *Environmental Science and Technology* 26(1):96-101.
- Edmunds, W.M., and A.H. Bath. 1976. Centrifuge extraction and chemical analysis of interstitial waters. *Environmental Science and Technology* 10:467-472.
- Emerson, K., R.C. Russo, R.E. Lund, and R.V. Thurston. 1975. Aqueous ammonia equilibrium calculations: effect of pH and temperature. *Journal of Fisheries Research Board of Canada* 32:2379-2383.
- Fitzpatrick, W.P., and N.G. Bhowmik. 1990. Pollutant transport to Lake Calumet and adjacent wetlands and an overview of regional hydrology. Illinois Department of Energy and Natural Resources, Hazardous Waste Research and Information Center, Champaign, IL. HWRIC RR-050. 74 pp.
- Giesy, J.P., C.J. Rosio, R.L. Graney, and M.G. Henry. 1990. Benthic invertebrate bioassays with toxic sediment and porewater. *Environmental Toxicology and Chemistry* 9:233-248.

- Harrison, W., E.T. Kucera, C. Tome, L.S. Van Loon, and A. Van Luik. 1981. Chemistry of bottom sediments from the Cal-Sag Channel and the Des Plaines and Illinois River between Joliet and Havana, Illinois. Argonne National Laboratory, Energy and Environmental Systems Division, U.S. Department of Energy. Contract No. W-31-109-Eng-38. 59 pp.
- Howes, B.L., J.W.H. Dacey, and J.M. Teal. 1985. Annual carbon mineralization and belowground production of *Spartina alterniflora* in a New England salt marsh. Ecology 66(2):595-605.
- International Joint Commission (IJC). 1988. Options for the remediation of contaminated sediments in the Great Lakes. Great Lakes Water Quality Board, Sediment Subcommittee, Windsor, Ontario, Canada. 78 pp.
- Illinois Environmental Protection Agency. 1990. Illinois water quality report 1988-1989. IEPA/WPC/90-160. Illinois Environmental Protection Agency, Division of Water Pollution Control, Springfield, IL. 352 pp.
- Jones, R.A., and G.F. Lee. 1988. Toxicity of U.S. waterway sediments with particular reference to the New York Harbor area. Pages 403-417 in J.J. Lichtenber, F.A. Winter, C.I. Weber, and L. Fradkin, eds. Chemical and Biological Characterization of Sludges, Sediments, Dredge Spoils, and Drilling Muds. American Society for Testing and Materials, Philadelphia. ASTM STP 976.
- Keeney, D.R. 1973. The nitrogen cycle in sediment-watersystems. Journal of Environmental Quality 2:15-29.
- Kleerekoper, H. 1953. The mineralization of plankton. Journal of Fisheries Research Board of Canada 10:283-291.
- Knezovich, J.P., and F.L. Harrison. 1988. The bioavailability of sediment-sorbed chlorobenzenes to larvae of the midge, *Chironomus decorus*. Ecotoxicology and Environmental Safety 15:226-241.
- Lubinski, K.S., M.J. Wallendorf, and M.C. Reese. 1981. Analysis of Upper Mississippi River System correlations between physical, biological and navigation variables. Technical Report in partial fulfillment of Contract No. 895-305. Upper Mississippi River Basin Commission, St. Paul, MN.
- Martell, F.L., R.T. Motekaitis, and R.M. Smith. 1990. Structure-stability relationships of metal complexes and metal speciation in environmental aqueous solutions. Environmental Toxicology and Chemistry 7:417-434.
- Mathis, B.J., and T.F. Cummings. 1973. Selected metals in sediments, water, and biota in the Illinois River. Journal Water Protection Control Federation 45(7):1573-1583.

- Mount, D.I. 1988. Methods of aquatic toxicity identification evaluations: phase III toxicity confirmation procedures. EPA Research Series Report. Environmental Research Laboratory, Duluth, MN. EPA/600/3-88/036.
- Mount, D.I., and L. Anderson-Carnahan. 1988. Methods of aquatic toxicity identification evaluations: phase I toxicity characterization procedures. EPA Research Series Report. Environmental Research Laboratory, Duluth, MN. EPA/600/3-88/034.
- Mount, D.I., and L. Anderson-Carnahan. 1989. Methods of aquatic toxicity identification evaluations: phase II toxicity identification procedures. EPA Research Series Report. Environmental Research Laboratory, Duluth, MN. EPA/600/3-88/035.
- Nebeker, A.V., G.S. Schuytema, N.L. Grffis, J.A. Barbitta, and L.A. Carey. 1989. Effect of sediment organic carbon on the survival of *Hyalella azteca* exposed to DDT and Endrin. Environmental Toxicology Chemistry 8:705-718.
- Neves, R.J., ed. 1987. Proceedings of the workshop on die-offs of freshwater mussels in the United States. U.S. Fish and Wildlife Service and Upper Mississippi River Conservation Committee. 23-25 June, Davenport, IA.
- Polls, I., S.J. Sedita, D.R. Zenz, and C. Lue-Hing. 1985. Illinois Waterway water quality survey from the Lockport Lock and Dam to the Peoria Lock and Dam 1977 and 1983. The Metropolitan Sanitary District of Greater Chicago, Department of Research and Development, Report No. 85-14. 220 pp.
- Reinbold, K.A., and S.M. Pescitelli. 1990. Effects of cold temperature on toxicity of ammonia to rainbow trout, bluegills, and fathead minnows. Illinois Natural History Survey Aquatic Ecology Technical Report Contract 68-01-5832/B. 15 pp.
- Ross, P.E., V. Jarry, and H. Sloterdijk. 1988. A rapid bioassay using the green alga, *Selenastrum capricornutum*, to screen for toxicity in St. Lawrence River sediment elutriates. Pages 68-73 in J. Cairns, Jr., and J.R. Pratt, eds. Functional Testing of Aquatic Biota for Estimating Hazards of Chemicals. American Society for Testing Materials, Special Technical Publication No. 988, Philadelphia, PA. 242 pp.
- Salomons, W., N.M. de Rooij, H. Kerdijk, and J. Bril. 1987. Sediments as a source for contaminants. Hydrobiologia 149:13-30.
- Schubauer-Berigan, M.K., and G.T. Ankley. 1991. The contribution of ammonia, metals and nonpolar organic compounds to the toxicity of sediment interstitial water from an Illinois River tributary. Environmental Toxicology and Chemistry 10:925-939.

- Serruya, C. 1974. Nitrogen and phosphorus balances and load-biomass relationship in Lake Kinneret. Verh. Internat. Verein. Limnol. 19:1357-1369.
- Snell, T.W., and G. Personne. 1989. Acute toxicity bioassay using rotifers II. A freshwater test with *Brachionus rubens*. Aquatic Toxicology 14:81-92.
- Sparks, R.E. 1977. Environmental inventory and assessment of navigation pools 24, 25, and 26, Upper Mississippi and Lower Illinois rivers. An electrofishing survey of the Illinois River. Special Report No. 5. UILU-WRC-77-0005. University of Illinois, Urbana-Champaign Water Resources Center. 122 pp.
- Sparks, R.E. 1984. The role of contaminants in the decline of the Illinois River: Implications for the Upper Mississippi. Pages 25-66 in J.G. Wiener, R.V. Anderson, and D.R. McConville, eds. Contaminants in the Upper Mississippi River. Proceedings of the 15th Annual Meeting of the Mississippi River Research Consortium. Butterworth Publishers, Stoneham, MA. 368 pp.
- Sparks, R.E. 1992. The Illinois River-Floodplain Ecosystem. Pages 412-432 in National Research Council. Restoration of aquatic ecosystems: science, technology, and public policy. National Academy Press, Washington, D.C. 552 pp.
- Sparks, R.E., M.J. Sandusky, and A.A. Paparo. 1981. Identification of the water quality factors which prevent fingernail clams from recolonizing the Illinois River, Phase II. University of Illinois, Water Resources Center Research Report No. 157. Urbana, IL.
- Sparks, R.E., and K.D. Blodgett. 1983. Toxicity of sediments in the Illinois Waterway: implications for the proposed increases in diversion of Lake Michigan water into the Illinois Waterway. Project Completion Report STIL-TRWRTOXSED39483, U.S. Army Corps of Engineers, Chicago, IL.
- Sparks, R.E., and M.J. Sandusky. 1983. Identification of the water quality factors which prevent fingernail clams from recolonizing the Illinois River, Phase III. University of Illinois Water Resources Center Research Report No. 179. Urbana, IL. 55 pp.
- Sparks, R.E., and P.P. Tazik. 1986. Des Plaines River long-term monitoring program: Phase I. Illinois Natural History Survey, Center for Aquatic Biology Technical Report 1986(6). 110 pp.
- Sparks, R.E., F.S. Dillon, and J.L. Arnold. 1992. Illinois River fingernail clam toxicity study. Final Report Project F-94-R-1. Illinois Department of Conservation, Springfield, IL.
- Starrett, W.C. 1972. Man and the Illinois River. Pages 131-169 in R.T. Oglesby, C.A. Carlson, and J.A. McCann, eds. River Ecology and the Impact of Man. Academic Press, NY.

- Swartz, R.C., G.R. Ditsworth, D.W. Schults, and J.O. Lamberson. 1985. Sediment toxicity to a marine infaunal amphipod: cadmium and its interaction with sewage sludge. *Marine Environmental Research* 18:133-153.
- Swartz, R.C., P.F. Kemp, D.W. Schults, and J.O. Lamberson. 1988. Effects of mixtures of sediment contaminants on the infaunal amphipod, *Rhepoxynius abronius*. *Environment Toxicology and Chemistry* 7:1013-1020.
- Swartz, R.C., D.W. Schults, T.H. Dewitt, G.R. Ditsworth, and J.O. Lamberson. 1990. Toxicity of fluoranthene in sediment to marine amphipods: a test of the equilibrium partitioning approach to sediment quality criteria. *Environmental Toxicology and Chemistry* 9:1071-1080.
- Talkington, Laurie McCarthy. 1991. The Illinois River: working for our state. Illinois State Water Survey, Miscellaneous Publication 128. Champaign, IL. 51 pp.
- Tessier, A., and P.G.C. Campbell. 1987. Partitioning of trace metals in sediments: relationships to bioavailability. *Hydrobiologia* 149:43-52.
- U.S. Army Corps of Engineers. 1987. Illinois River charts. U.S. Army Corps of Engineers.
- U.S. Department of Commerce and Labor. 1911. Special reports. Fisheries of the United States 1908. U.S. Government Printing Office, Washington, D.C. 324 pp.
- U.S. Environmental Protection Agency. 1985. Methods for measuring the acute toxicity of effluents to freshwater and marine organisms. Environmental Research Laboratory, Cincinnati, OH. EPA/600/4-85-013.
- U.S. Environmental Protection Agency. 1988. Toxic sediments: approaches to management. U.S. Environmental Protection Agency, Science, Policy Integration Branch, Washington, D.C. EPA 68-01-7002. 90 pp.
- U.S. Environmental Protection Agency. 1992. Toxicity identification evaluation: characterization of chronically toxic effluents. Phase I. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/6-91/005. 73 pp.
- Wilson, D.M., T.J. Naimo, J.G. Wiener, R.V. Anderson, M.B. Sandheinrich, and R.E. Sparks. (Submitted). Declining populations of the fingernail clam *Musculium transversum* in the Upper Mississippi River. Submitted to the Journal of the North American Benthological Society.

REPORT DOCUMENTATION PAGE		1. REPORT NO. ILENR/RE-WR-92/07	2.	3. Recipient's Accession No.
4. Title and Subtitle Identification of Toxic Substances in the Upper Illinois River			5. Report Date October 1992	
7. Author(s) Richard E. Sparks and Philippe E. Ross			8. Performing Organization Rept. No.	
9. Performing Organization Name and Address River Research Laboratory Forbes Biological Station Illinois Natural History Survey P.O. Box 599 Havana, IL 62644			10. Project/Task/Work Unit No. 89/215	
12. Sponsoring Organization Name and Address Illinois Department of Energy and Natural Resources Office of Research and Planning 325 West Adams Street Springfield, IL 62704-1892			11. Contract(C) or Grant(G) No. (C) (G) WR3691	
15. Supplementary Notes			13. Type of Report & Period Covered	
16. Abstract (Limit: 200 words) Between 1955 and 1958, several abundant species of aquatic insects, snails and finger-nail clams practically disappeared from the upper Illinois River. These declines had equally drastic repercussions on the populations of ducks and fish that fed upon these invertebrates. The situation changed very little into the 1980s, despite improvements in water quality. This research found that porewater from relevant river sediments contains a toxic factor that inhibits the filtering ability of fingernail clams and also negatively effects the water flea, while stimulating alga and bacteria growth. All evidence points to ammonia as the toxic agent. Also, at two sites, the porewater contained toxic oil products, including polycyclic aromatic hydrocarbons, such as naphthalene. A three phase Toxicity Identification and Evaluation methodology was utilized in reaching these conclusions.			14.	
17. Document Analysis a. Descriptors Water Pollution, Toxicity, Water Pollution Effects (Animals), Sediments, Sedimentation, Suspended Sediments				
b. Identifiers/Open-Ended Terms Sedimentation - Illinois River Toxicity - Illinois River Water Pollution - Illinois River				
c. COSATI Field/Group Biological and Medical Sciences; Environmental Biology				
18. Availability Statement No restriction on distribution. Available at IL Depository Libraries or from National Technical Information Service, Springfield, VA			19. Security Class (This Report) Unclassified	21. No. of Pages 72
			20. Security Class (This Page) Unclassified	22. Price

